A TEXT BOOK
OF
PLANT PATHOLOGY

By
EDGAR F. VESTAL

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Plant pathology..

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A TEXT-BOOK OF

PLANT PATHOLOGY

DEALING WITH THE MAJOR DISEASES OF THE
MORE COMMON NORTH INDIA CROP PLANTS

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By

EDGAR F. VESTAL

BOTANIST AND PLANT PATHOLOGIST
ALLAHABAD AGRICULTURAL INSTITUTE

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PREFACE

The material presented in this book has been collected over a period of some ten years during which time the author was a member of the staff of the Agricultural Institute, Allahabad, India. The book is the outgrowth of "Notes on Plant Pathology" printed in 1941 and reprinted, with some alterations, in 1946.

Chapters 1 to V inclusive include the more or less fundamental topics, such as, classification, agents of plant disease control, methods of plant disease dissemination and the common fungi associated with plant disease. Chapters VI to XIV deal with the more important common crop plant diseases found in northern India as well as some of the more important ones of other sections. Not all of the known diseases are included and some may disagree with the selection of material presented. The Allahabad Agricultural Institute receives students from all parts of India and there is a continual request from them for information about some of the diseases of their part of the country. This accounts for some diseases being discussed that are not common in the United Provinces or areas with a similar climate.

A few diseases are presented that are not fully known. Examples are the anthracnose and *Alternaria* of alfalfa. They are presented here with such information as was at hand with the hope that before long the whole life cycle and control may be known.

The drawings and photographs are submitted as guides and not as works of art. They are merely as guides to the student for one of the first things looked for is an illustration. Unfortunately not all of the diseases have been illustrated. Illustrations presented are all from local material.

An earnest effort has been made to limit all errors to mechanical errors. That this will be found correct is hardly likely but it is sincerely hoped that such as are found will be mechanical and not errors of fact. The author will appreciate it if his attention is called to any serious error of fact that they may be corrected before another edition is made.

The book in its present form and shortcomings is a test of ideas and its practical usefulness will be known after it has been put to a good test.

Appreciation and thanks are due to the members of the staff of the Agricultural Institute who have offered suggestions and criticisms. To Dr. J. L. Goheen for providing clerical aid for portions. To the Standard Agricultural Chemicals, Hoboken, New York, for the pictures shown in Chapter III. To Dr. B. B. Mundkur for kindly criticisms and for his permission for use of the list of Indian smuts at the end of Chapter VII. To Mr. K. B. Pisharodi for various illustrations and to Mrs. Vestal for reading, correcting and typing several of the Sections. Especial appreciation is due to Mrs. Vestal for patiently enduring the ordeal of having her husband spend so much of his spare time in the preparation of the manuscript.

EDGAR F. VESTAL
January 1948

NOTE

The author of this text left India before the proof of the manuscript could be prepared. This necessitated sending the proofs across the ocean to Central America for reading. Because of the time consumed in the mail it seemed advisable to arrange for a major portion of the reading to be done by some one at the Allahabad Agricultural Institute. Dr. T. A. Koshy, who received his Ph.D. in plant pathology at the Ohio State University, Columbus, Ohio. U. S. A., in 1948, was well qualified to do the reading. Dr. Koshy kindly consented to do the reading and it is to him that much credit is due for the elimination of errors and final arrangement of materials in the book.

The author wishes to express his deep appreciation and gratitude to Dr. Koshy for assuming the role of joint editor for the book.

EDGAR F. VESTAL, Research Professor,
Iowa State College—Guatemala,
Tropical Research Center,
Antigua, Guatemala.

INTRODUCTION

The introduction is separated into two sections in the hope that by so doing the student may be given a better idea of the field of plant pathology. The short historical review is to give the student a glimpse of the development of plant pathology and the section on parasitism is to give him an idea of the problems facing the plant pathologist in dealing with the plant and its pathogens.

HISTORICAL REVIEW

The study of plant pathology is a relatively new science. If we turn back 100 years in the history of the world we would find ourselves in the period before any plant pathology had been developed. By 1848 there had been little real work done along the line of associating living organisms with plant disease. Louis Pasteur had just begun his work and it was at least three decades before his contributions would become accepted by the world. Darwin was soon to create a stir with his theory of the origin of species. But the three decades after 1848 saw relatively little contributed toward the knowledge of plant disease.

In 1878 there were two events that, while they had no relation to each other, were landmarks in the progress of the thinking of the time. Sir Joseph Lister performed the first operation under aseptic conditions, using phenol spray, and Professor Burrell, of the University of Illinois, first associated bacteria with plant disease. Dr. Lister's work was acclaimed with great thanksgiving while that of Professor Burrell was challenged by the plant pathologists of Europe. Dr.

Erwin F. Smith took up the challenge of Prof. Burrell's work and for some years there was a verbal battle carried on over the possibility of bacteria living under the conditions then believed to exist in a plant body. Plant sap was then thought to be acid and bacteria were held to be able to live only under alkaline conditions. We now know the outcome of that debate.

It was during this early period that Anton De Bary, the great German worker, made a critical study of many fungi, including the black stem rust of wheat and the late blight organism of potato. It was during this period that Bentham and Hooker were publishing their floral studies in India.

During these early days sulphur was unknown as a fungicide. Arsenicals were not used for insects and such gases as carbon disulfide were unknown. There were no periodicals published which had any section devoted to plant disease control. But the period from 1878 to 1890 was filled with startling discoveries. The works of Burrell and Lister have already been referred to. The discovery of the organism causing leprosy, those causing glanders, typhoid fever, pneumococcus and diphtheria between 1880 and 1884 were not actually plant pathological but they did much to stimulate the study of plant disease. In England progress was being made. Marshall Ward went to Ceylon to study the diseases of the coffee plant which were causing losses for the decade, totalling some 15,000,000 pounds sterling.

In 1882 Saccardo began his great work "Sylloge Fungorum". By 1882 the first book of plant disease appeared when Hartig published his "Lehrbuch der Baumkrankheiten". In 1883 the "Berichte d. d. botanischen Gesellschaft" was started and the first real botanical journal was on its way.

Millardet made his recommendations for the use of Bordeaux mixture in 1885. Tobacco mosaic was described by Meyer in 1886. In 1886 Sorauer published the second edition of "Pflantzenkrankheiten" which was a mine of information for the workers of that time. For the next few years the reports of the discovery of plant diseases appeared in rapid fire order like those of animal and human diseases during the first part of the decade. In 1887 the Annals of Botany began being published. The journal of Mycology began in 1889. The Centralblatt "fur Bakteriologie" began in 1895. In 1895 Wakker and Went came out to Java to study the cane diseases of the Islands. It was during the 1880—1890 period that the *Phylloxera* of grapes was conquered in France with the use of flooding, insecticides, and, grafting into resistant stocks. It was the first real triumph of man over the pests which were destroying his crops.

In 1896 mercuric chloride came into use as a seed treatment. It was used for treating for potato scab. It was during this decade, 1890—1900 that saw the coffee plants replaced by tea because of the uncontrolled ravages of the coffee rust *Hemileia vastatrix*. It was during this period that it was pointed out animal and plant diseases could be disseminated by insect and other agents. New discoveries came so fast that the field of investigation seemed to literally explode. Curcubit wilt was described and the agent of transmission, *Diabrotica vittata*, the cucumber beetle, was demonstrated. It was demonstrated that the bacterial brown rot of potato was transmitted by the Colorado potato beetle.

On the animal side, symptomatic anthrax was described. Roux and Yersin found the organism of diphtheria and, also, that it produced in the body of the victim an antitoxin. Koch produced the tuberculin which was hailed as a great cure for the dreaded T. B. Behring and Kitasato developed tetanus antitoxin. Amoebic dysentery was demonstrated for the first time. Yersin and Kitasato demonstrated the organism of plague. Serum anaphylaxis was first shown. By the beginning of this century some 22 groups of bacteria had been recognized of which at least 16 contained pathogenic forms.

In 1900 L. R. Jones published his critical study of the soft rot organism of carrots. The same year Bailey published the Encyclopedia of Horticulture. J. C. Arthur began the study of rusts which was to finally result in the publication of the splendid book "The Manual of Rusts of the United States and Canada" (31) Migula's second edition of "System der Bakterien" appeared. Bolley described the cause of flax wilt and assigned the cause to *Fusarium lini. Rhizoctonia* of potatoes and root rot, caused by *Fusarium oxysporium*, were described. Black leg of potatoes was considered to be caused by *Bacterium phytophthora.* Stem rot of coffee was attributed to *Stilbella flavida.*

So it went. To make a complete list of all of the diseases would be outside the scope of this book and would likely bore most of the readers. But enough has been given to indicate the importance of the period.

The period 1900 to 1910 saw a number of advances, perhaps the most important of which would be the publication by Appel and Wollen-waber of the "Genus *Fusarium*". This monograph is still looked to as the real authority with regard to the Fusarium species. B. M. Duggar published the first American text-book on plant pathology in 1909.

From 1910 to 1920 was a year that saw some very important events happen. Sulfuric acid was used for the first time as a control of damping off and the first disease resistant plant was produced by plant breeding. Norton produced a rust resistant asparagus plant. In 1913 Stevens wrote "Fungi which Cause Plant Disease". In 1915 Jones and Gilman demonstrated that *Fusarium* diseases could be controlled by breeding resistant varieties when they produced a *Fusarium* resistant cabbage. It was during this period that the work on viruses was given a start. The study of bacteriophage was also undertaken during this decade. "Fungi and Disease in Plants" by E. J. Butler was published in India.

Between 1920 and 1930 further changes appeared in the point of view of the plant pathologist. It was during this period that the idea of strains became prevalent. Such things as physiologic forms or races were soon considered beyond the controversial point. Koch's idea of the fixity of species was to receive many a rude shock before the end of the decade. Over in Java Miss Wilbrink demonstrated that the disease of sugar-cane, known as the Sereh disease, could be controlled by heating the canes with water at 45° C. for 30 min. then heating at 50° to 53° C for 30 min.

From 1930 to the present time has seen a new science spring up. Progress has been perhaps even more spectacular although we may not have been able to be such close observers. It has been during this period that, over the world, the emphasis has been along the lines of plant breeding. Hybrid corn was first produced commercially. Rust resistant varieties of wheat, oats and barley were produced in many countries. Plant pathologists turned from the use of chemical dusts and sprays to the production of resistant varieties as a means of control of disease. However, not all of the emphasis was placed on plant breeding by any means for the commercial companies had too much money invested in factories and personnel to give up so easily. New types of machines were soon produced and the age of power machines was a reality. Dusts were introduced as a substitute for the bulky, messy water sprays. The big commercial companies set up their own experiment stations and hired the best trained college graduates available to work for them. That this programme paid out was amply demonstrated during World War II when the commercial company laboratories were the backbone of the defence of most of the nations, certainly of the U. S. A.

Today we see the plant breeder on the one hand working to develop resistant varieties of plants for those diseases that are not readily controlled by sprays or dusts. We see also the commercial companies with their well-equipped laboratories and skilled staff testing machines and chemicals for every type of condition imaginable and then demonstrating to the farmer how to use them. We find the use of inorganic and organic manures receiving the attention of the farmers for the control of some of the soil borne plant diseases. Looking still further into the future, the world may some day see plant disease controlled by the use of hormones or growth inhibiting substances. One thing is certain, we are not yet at the end of the discoveries along the line of plant disease control.

PARASITISM

Plant pathology deals directly or indirectly with the subject of parasitism. That being the case it might be well for us to stop a moment and think over the subject. What constitutes parasitism? A dictionary definition is not satisfactory for it is too indefinite. We then turn to the scientists for the definition but find that they are not always in agreement as to the definition. The diversity of opinion is no doubt due to the fact that scientists approach the subject from so many different angles. For the most part the term parasitism is very much less used than the terms "resistance" or "susceptibility." Most of the recent literature takes up the subject of disease resistance but say comparatively little about parasitism itself.

T. F. Yu (1941) stated that parasitism consists of three stages: *i.e.*, (*a*) growth of the parasite outside of the host plant; (*b*) entrance of the parasite into the plant; (*c*) the establishment of parasitism inside the host plant. Perhaps this might be considered a roundabout way of defining parasitism. That is, parasitism is a condition of antagonistic symbiosis in which there is injury to one symbiont by the other. The state before parasitism has been discussed by Brown (88). He shows that many plants give off electrolytes which definitely affect the germination of spores in contact with the epidermal surface of the host plant. He also mentions that certain volatile substances are given off which accumulate in the air surrounding the leaf or stem surface and influence the germination of spores. The idea is suggested by the fact that certain diseases appear most often on certain parts of the host plant, indicating that conditions for growth are most suitable on these parts. Morphology of the host plant may play a part for such parts, as the petals and other flower parts, are much more readily attacked by fungi than the more resistant leaves or stems. However, it is held that, in addition to the favourable morphological structures found in the flower, the floral parts excrete certain electrolytes which aid the fungus spores to become established.

For the parasite to be able to live outside of the host plant certain factors must persist. Food, optimum temperature and humidity must be present. In addition there must be an opportunity for the pathogen to become a pathogen. In other words there must be freedom of action. Recently there has been a good deal of discussion on the subject of antagonism or competition between fungi. In 1938 Porter and Carter (617) presented a very interesting review on this subject. They state that many fungi give off substances that are distinctly harmful or distinctly beneficial to other fungi. Yu (941) makes the observation that any factor which destroys or inhibits any one of the three stages mentioned, destroys parasitism. Thus the stimulation of saprophytic fungi, which are antagonistic to the pathogens, by the use of organic manures, is one means of breaking the parasitism of root rotting plant pathogens. Thus destruction of the pathogen outside of the host plant may be accomplished. The saprophytic fungi destroy the spores, mycelium, sclerotia and other fungal masses before they have an opportunity to gain entrance to the host plant.

The second phase of parasitism as stated by Yu is the gaining of entrance into the host plant. This depends upon several factors. Already mentioned are, food, temperature and humidity. To these may be added soil reaction and the condition of the host plant. Soil reaction is a factor in many cases. Garrett (242) discusses the soil reaction and offers some interesting data bearing on this subject. He states that some fungi are inhibited by the accumulation of carbon dioxide in the soil and that alkaline soils were favourable for fungal growth because they acted as acceptors of the CO^2 which is given off by the roots. Garrett (Chapter 6 pp. 54-60) mentions numerous cases in which he states that the concentration of plant nutrients has reduced the incidence of plant disease. This appears to be partly due to the influence upon the pathogen and partly an influence upon the host. Not all plant food elements behave alike with regard to root rot fungi. The statement, made formerly by numerous plant physiologists, that nitrogen predisposes toward disease, is not always true and has recently been revised. Experiments have shown in a number of cases that the nitrogen content of the soil and root rotting, of such plants as cotton, are negatively correlated. At the same time other workers, using other crops, have found that a low nitrogen content with a high phosphorus content, has reduced *Pythium* root rot of cereals. Potash added to the soil has offered some control over Fusarium vasinfectum, the wilt of cotton. But the plant pathologists are not yet agreed as to the exact explanation of the control, or the lack of control as the case may be, through the use of fertilizers. That is, is the effect of the nutrient upon the host, parasite, or both?

Another factor affecting the entrance of the pathogen into the host is its ability to penetrate the host plant tissues. In many cases penetration is made by way of the stomata. In others by wounds. Some spores are able, by means of appresoria, to directly pierce the epidermal tissues and thus gain entrance. In the case of some a secretion is made by an agent in dissolving the host tissues so that the pathogen may gain entrance.

On the part of the host plant there may be some characters that aid it to ward off the pathogen. In some cases the morphology is such that the pathogen can enter only with difficulty. Or, if entrance is gained, the invader may not be able to establish itself or to reproduce itself. An example may be given of the Hope wheat, developed in the United States, which possesses a very strong stem. When this wheat is infected by stem rust the rust sori are unable to rupture the epidermis and thus the spores are prisoners. This is a case of morphology as the sclerenchyma is much more developed in the Hope wheat than other varieties.

Yu's third point has to do with the establishment of the parasite within the host. Much of the recent discussion on this phase of disease resistance has centred around the genetical behaviour of the plant. Wingard (933) has discussed this phase and the student should read his article in the Botanical Review. The hereditary nature of disease resistance is common knowledge but the disconcerting thing about disease resistance is that it does not follow any single law. Results have been extremely diverse. In some cases they could be explained on a single factor hypothesis while in others it required a multiple factor hypothesis to account for the various reactions. Disease resistance is often found in the wild counterparts of the commercial plants, which are themselves, of no commercial value. The factors must be combined with good commercial factors to be of economic value. An example which illustrates the point is that of watermelon wilt. The disease is caused by *Fusarium niveum* and against which no commercial varieties showed any resistance. By crossing the African Stock Citron with one of the best commercial varieties it was possible to secure a resistant hybrid in the F_1. In this case susceptibility was dominant (560). Tisdale (817) found that resistance to flax wilt, (*Fusarium lini*), was based on a multiple factor hypothesis and he secured all degrees of resistance. Walker (915) has written an excellent article on disease resistance in vegetable crops which is worth reading. He notes that the hypha of many fungi enter the stomata of plants other than their host plants. This has been noted in a number of cases. But the fungi could not survive. In some cases there has been direct penetration of the host plant tissues but the fungus could go no farther. It was unable to establish itself after gaining entrance.

Why is the would-be-parasite not a parasite? Explanations are incomplete. It is one thing to say that there is an incompatibility between the fungus and the invaded plant but what does it mean? In some cases it may be lack of certain elements needed by the fungus. Garrett (242) mentions the work of Spencer and McNew who found that the bacterial wilt of maize, (*Bacterium stewarti*) was much worse on plants that had received an excess of nitrogen than on plants grown in nitrogen deficient soil.

Variability of the host and parasite present many knotty problems to the plant breeder. Mutations among the fungi occur and this has meant a constant menace to the worker who may have spent years perfecting a certain resistant plant and has visions of it being completely useless if a mutation should occur or a new disease appear. At this writing, Dec. 1947, this very thing has happened to the breeders of rust resistant oats in the United States. While they were having phenomenal success in breeding rust resistant oats and were rapidly replacing the old varieties with the new improved ones, a disease, caused by *Helmin-*

thosporium victoriae, suddenly appeared and threatened to undo all of the previous work. Breeders in the state of Iowa, and neighbouring states in the U. S. A. have set about to secure resistant hybrids. Fortunately some already exist, but in the meantime while these are being substituted for the susceptible ones, the disease threatens to become one of the most serious for some time.

As the pathogen passes from one susceptible host to another its virulence may be increased and an epiphytotic, that was mild in the beginning, may become extremely severe as the season progresses. On the other hand passing through a host plant may reduce the virulence of a pathogen and what was a virulent form of the disease may become even mild before the end of the season.

In some cases penetration has been associated with the secretion of an enzyme. Brown (88) states that there is no correlation between enzyme secretion in some cases and ability of the pathogen to parasitize the host. He goes on to state that we do not know the mode of activity of the enzyme and until we know more of that we cannot hope to understand the phenomena of parasitism. Walker (915) has given an excellent list of the vegetables and their disease reactions but he does not venture an explanation of the phenomena. It is evident that much is left to be learned before we shall be able to give a satisfactory explanation to the questions on parasitism, or, as was stated earlier, disease resistance in plants.

CHAPTER I

THE COMMON FUNGI ASSOCIATED WITH PLANT DISEASE

The fungi are a part of the division *Thallophyta*, which develops no true root, stem or leaf, but instead, develops a thallus body. This is one which has no real organization such as leaf, stem, root, flower, etc, The thallus body may vary from a single cell to one that is composed of a mass of thread-like hyphae. The thallus is devoid of chlorophyll although it may possess other forms of colouring matter which may range from gray to bright reds and blues.

The fungi absorb their food from the surrounding medium for they have no power to manufacture food of their own. Thus the fungus parasite must live at the expense of the host plant upon which it feeds. For the most part the farmer is concerned with those that feed upon the farm crop plants.

The effect of parasites upon the host plant.

In general any adverse effect upon the host plant is termed disease. A definition of disease might help to make clearer what is meant. Dr. L. R. Jones, formerly Head of the Department of Plant Pathology of the University of Wisconsin, U. S. A., defined disease as, "any departure from the normal form of function." What is normal is not always easy to explain or illustrate. What is normal under one set of conditions may not be normal for another and what is normal for one plant may not be normal for another. Thus before one can, with certainty, describe something as abnormal, the normal for that condition must be understood. When we talk of plant disease we must be certain that we understand the plant about which we are talking.

The effects upon the host plant may be varied and many. One of the most common symptoms of disease is a change in the chlorophyll pattern. Usually this is a loss of the green colour, although, in the case of some virus diseases, the colour may become a deeper shade. The virus diseases are among the most common that are associated with a lowering of the green colour in plants. Wilts and root rots are also associated with a yellowing of the leaves, as is insect injury.

Necrosis (death) of the host tissue is also a symptom of fungus attack. Necrosis may occur in association with numerous other disease symptoms but the amount will vary with the type of disease and the severity of the attack. A common type of necrosis is the leaf spotting of such fungi as *Cercospora*, *Alternaria* and *Helminthosporium*. Early blight of potatoes and the tikka disease of groundnuts are other examples. Necrosis may be in the form of a soft rot, as in the bacterial rot of vegetables; the leaves may become soft and discoloured, as in the case of late blight of potatoes, or the branches may be woody and the dead tissue be hard and rough as in the case of die back of *Citrus*. Root necrosis is a very common result of the activity of such fungi as *Fusarium*, *Pythium* and *Rhizoctonia*. The symptoms of root necrosis are usually a yellowing of the leaves, a dropping of the fruit and stunting of the plants, followed by death.

Wilting is another of the symptoms of fungus activity. Wilting may or may not be associated with root necrosis. Wilting may be the result of a plugging of the vascular tubes, as in the case of the vascular wilt of cucurbits, the *Phytophthora* stem rot of *Piper betle* and others. In these diseases the wilting occurs at points several feet from the place of invasion by the pathogen. The wilting is thus a secondary rather than a primary reaction. In the case of the papaya root rot disease the roots are completely destroyed and thus no food or water may pass to the stem and leaves.

Damping off is the result of a rapid destruction of the plant tissues just at the surface of the ground. This may be caused by a number of fungi but the most common fungi to cause damping off are *Pythium de baryanum*, *Fusarium*, *Sclerotium*, *Rhizoctonia* and occasionally such fungi as *Cercospora* and *Helminthosporium*. Damping off is found mostly among the seedlings and is not usually considered as a disease of older plants. Damping off of rice seedlings was observed on the Institute farm one season and was considered to have been caused by *Piricularia oryzee*, that being the only fungus observed.

Early maturity is another symptom of disease. In some diseased plants the small number of fruits which are set usually mature faster. That is a rule of nature. In the orchard one will often find the diseased guava tree with small yellowing fruits that are not normal. The same is true of papaya, the apple and pear.

Malformation of parts is another symptom of disease. Malformation may be the result of the action of insects and animals as well as plants. Viruses also are likely to produce some form of distortion, stunting or even enlargement of parts. Common examples of distortions produced by fungi are the galls and pustules of the white rust of *Crucifers*, the distorted flowers of the mustard caused by *Peronospora parasitica* and the galls formed by corn smut and by *Protomyces*. Galls on the roots are formed by the action of nematodes and galls on many plant stems and leaves are the result of insect injury.

Types of Organisms Causing Plant Disease

If we adhere strictly to the definition set forth above, then we will need to list, in addition to fungi, all things that produce abnormal conditions. Such will include animals, birds, man and any other agent capable of producing disease.

Animals that cause plant disease

Among the animals would be rodents, birds, *Arthropoda* (mites, insects, millipeds), *Mullusca* (snails, slugs, etc.), *Nemethelminthes* (eel worms) and perhaps the *Annelida*. Such animals are not usually treated in a course of plant pathology but are more likely to be found in a course in economic zoology or entomology.

Parasitic Flowering Plants

cuscuta

These are plants which, like doddar, *Orobanche* or *Loranthus*, are either without chlorophyll or possess only a small amount and therefore must depend upon some chlorophyll bearing host for food. They feed upon the host plant by means of a foot-like root which is forced into the stem until it contacts the phloem vessels from which it takes the prepared food materials, thus robbing its host. Most farm boys are familiar with the yellow, thread-like vines of the doddar which are seen on hedges and even in the field on alfalfa and other legumes. In the tops of mango trees one may see a cluster of short branches bearing dull green leaves. This is likely to be the *Loranthus*. At the foot of such garden plants as the brinjal, tomato or turnip one may see a more or less naked flower stalk, varying from a few inches to a foot in height, with flowers that are often slightly purplish in colour. These are flower stalks of *Orobanche*, or dutchman's pipe. It is one of the most serious pests of the plants mentioned and when they are grown on the same soil year after year the parasite may become a limiting factor.

Algae and Lichens

Algae, such as *Cephaleuros*, may interfere with photosynthesis, but it is doubtful if there is any real injury caused to the plants. Rusty red spots are common on mango leaves and on tea leaves but probably do not really injure the plants.

The same might be said for the lichens. They do not injure the plants on which they grow but more than likely are epiphytes. However, it is generally considered that the lichens and mosses do not grow upon healthy-growing trees. Their appearance usually indicates a weakened condition of the host plant.

Bacteria

These are the causes of many plant diseases. The most common are the soft rots, the wilts, leaf spot diseases and bud rots. The soft rot organisms act upon the middle lamella of the plant tissues and thus loosen the cement that binds the cells together. When this occurs the plant cells lose moisture into the intercellular spaces and, as there is no support for the cells, they assume a rounded shape and the tissue becomes a soft watery mass usually with a disagreeable odour. There are a number of soft rot producing bacteria. *Bacterium carotovora* and *Bacterium phytophthora* are examples. The former is found on many vegetables and the latter principally on such crops as potato, radish, carrot, tomato and some of the legumes.

In some cases the stems are attacked above ground and a definite rot produced. Bacterial stalk rot of maize is an example. Stewart's disease of sweet corn is an example of stalk and leaf infection. In this type of disease the fields are uneven with stunted plants here and there. Larger plants exhibit water-soaked stripes in the leaves. When stems of diseased plants are cut across, drops of bacterial ooze come out. In fact the appearance of the drops of ooze is a symptom of a number of bacterial diseases.

Bacterial diseases are found on mango, maize, coconut, citrus, potato, beans, papaya, and many other plants. They are usually water and insect distributed but other agencies also play a part. Some are seed borne. Examples of seed borne bacterial plant pathogens are *Bacterium campestre*, the vascular disease of cabbage; *Bacterium stewarti*, already referred to above; *Bacterium phaseoli*, found widely distributed on legumes; *Bacterium malvacearum*, found on cotton.

Perhaps the most important characters to look for in a bacterial plant disease are the water-soaked appearance of the active areas and drops of ooze that may appear on the areas either before or after breaking the tissues. Neither of these may appear, so that it is not possible to identify positively a bacterial disease simply from a macroscopical observation, it may require microscopical examination and isolation in the laboratory.

Bacteria are round, rod or spiral in shape. They are formed singly, in chains or in clumps. They are non-motile or motile by means of flagella at one end, flagella at both ends or flagella all around the cell. (See under classification). Reproduction is by simple fission or by fragmentation. Under certain circumstances the contents of the cell round up, enclose themselves in a thick wall and become a resting spore. This is able to withstand extremes of heat, cold and drying and thus carry the bacteria over adverse conditions.

THE FUNGI

Myxomycetes

The *Myxomycetes* possess very little real organized structure. They are listed among the true fungi because they have in a part of the life cycle, structures that resemble the sporangia of some of the higher groups. They are often listed outside the true fungi because a part of the life cycle consists of little more than a naked mass of protoplasm (plasmodium). The plasmodium is multinucleate, of the consistency of the white of an egg, and moves about by a flowing motion, as an amoeba.

Two reproductive stages exist. The vegetative stage consists in the production of asexual spores simply or on complex fruiting bodies (sporangia) which may be very elaborate in some cases. The colours of the vegetative bodies may be as varied as the shape and structure. On some they are drab, gray or black, while on others they are various colours of violet, yellow or brown.

Under some conditions the spores germinate to form swarm spores (motile spores), or they may form naked amoeboid cells which may reproduce by simple fission, or they may fuse to form amoeboid masses (plasmodia).

The slime moulds are not usually destructive to cultivated plants. A considerable number have been recorded on dead wood and other parts of plants but most of them are evidently only saprophytes.

The Phycomycetes

The *Phycomycetes* are often referred to as the downy mildews because of the appearance of the mycelium. There are few cross walls to be seen in the mycelium of the *Phycomycetes* and for this reason they are referred to as possessing coenocytic mycelium. These are often referred to as the algae-like fungi from the resemblance to *Vaucheria*, green algae, that are also without cross walls. It is suggested that the student read the chapter on the *Phycomycetes* in *The Lower Fungi* by Fitzpatrick (226).

Aside from the mycelium, the typical asexual structure of the *Phycomycetes* is the sporangium. The shape and size of the sporangium varies with the different species and genera. See Plate No. I. The illustrations of *Saprolegnia*, *Rhizopus* and *Pythium* repre-

PLATE I

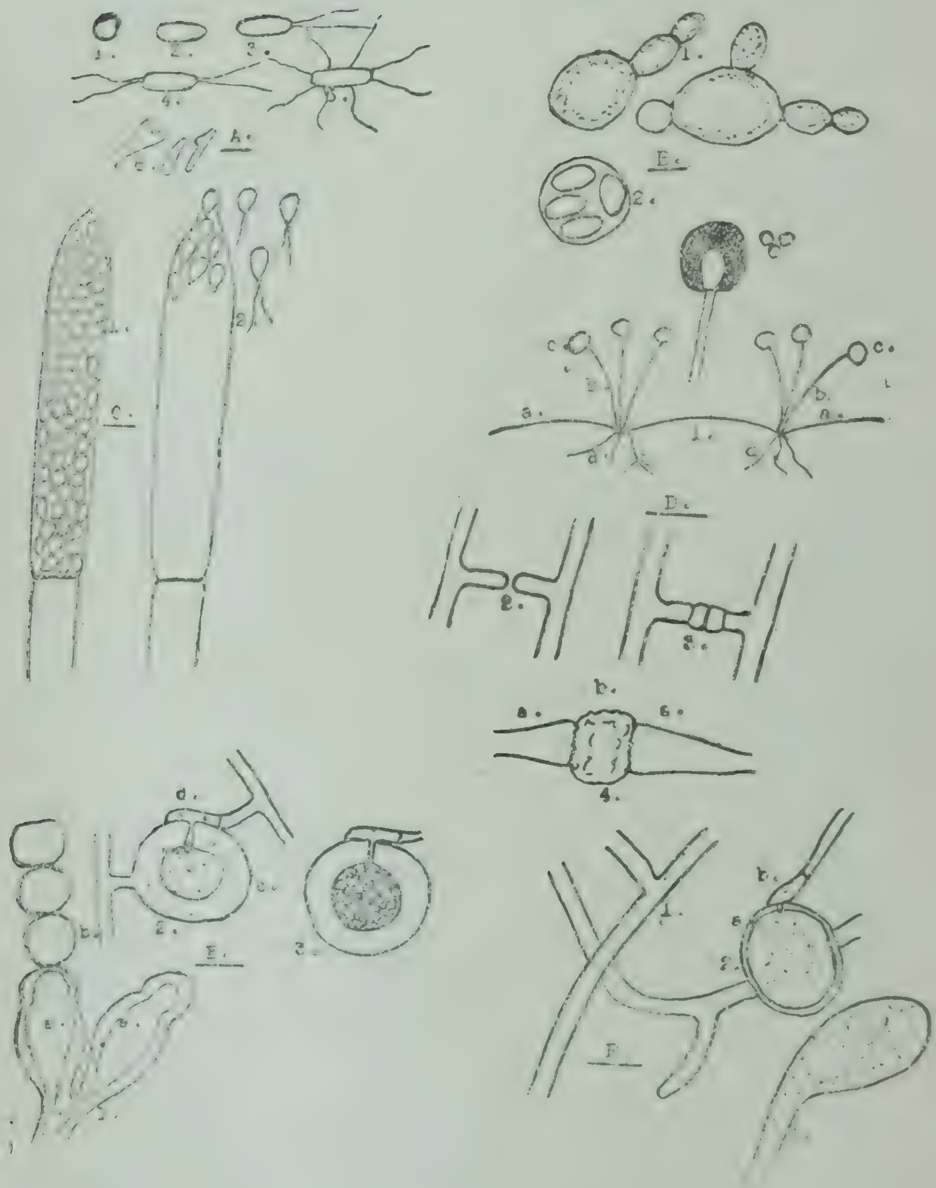

Plate No. I

A. Types of bacteria.

1. Coccus.
2. Rod.
3. Rod with polar flagella at one end.
4. Polar flagella at both ends.
5. Flagella all around (peritrichous).
6. Legume bacteria.

B. Yeast.

1. Simple budding.
2. Ascus with four ascospores.

C. *Saprolegnia.*

1. Sporangium with zoospores forming.
2. Partly empty sporangium with swarm spores outside.

D. *Rhizopus.*

1. Sketch of vegetative body. a. Stolon. b. Sporangiophore. c. Sporangium. d. Rhizoids.
2. First step in gamete formation.
3. Gametes formed and in contact.
4. a. Suspensors. b. Zygote.

E. *Albugo* (*Cystopus*).

1. Asexual reproduction. a. Conidiophores. b. Conidia.
2. Sexual reproduction. c. Oogonium. d. Antheridium.
3. Zygote. Outer oogonial wall and old antheridium still attached.

F. *Pythium.*

1. Coenocytic hyphae.
2. Sexual reproduction. a. Oogonium. b. Antheridium.
3. Sporangium.

sent the extremes of variation but between these are all gradations of shape and type.

The asexual spores are either motile or non-motile. Most of the *Pythiaceae* (see classification) are motile at one stage. The other members of the Class are non-motile. They are relatively short-lived but because they are produced in enormous numbers they are very important in the spread of the fungi.

The sexual phase of the *Phycomycetes* varies with the sub-class. In the sub-class *Oomycetes,* the female reproductive body, the oogonium, is typically a large, round, thin-walled cell, containing one or more nuclei and a large amount of nutritive material. The male organ, on the other hand, is small, more or less elongated, contains one or more nuclei and a small amount of cytoplasm. Fertilization is usually accomplished by means of a small tube (fertilization tube) which penetrated the oogonial wall and the cytoplasm far enough to permit the contents of the antheridium to pass over into the oogonium. (See Plate N. I, E). The fertilized oogonium is known as the zygote, or zygospore, and is usually a thick-walled resting spore. These spores are capable of withstanding extremes of heat, drouth and cold and are the main factors in perpetuating the fungus through adverse conditions. In some cases the zygospore germinates to form a mycelium directly, while in other cases it germinates to form a zoospore and then a mycelium.

Among the *Phycomycetes* are some of the most serious of the plant pathogens. Some of the common diseases caused by them are damping-off, caused by *Pythium debaryanum;* foot rot and leaf spot of pan, caused by *Phytophthora parasitica;* late blight of potatoes, caused by *P. infestans;* white rust of crucifers, caused by *Albugo candida;* the downy mildew of bajra and maize, caused by *Sclerospora, grammicola;* and the downy mildew of alfalfa caused by *Peronospora trifoliorum.*

The term "downy mildew" is intended as a descriptive term for the appearance of the disease plants. The symptoms vary but in general they are first observed as a lighter shade of green colour that changes into a yellow. The spots may bear a water-soaked appearance and there may be dwarfing, deforming and killing of the parts infected. In some cases, as for example, the white rust, the tissues are enlarged into galls. Necrosis usually follows attacks and the loss is often severe. The dwarfed and barren stalks of bajra are a common sight in the fields and it is not uncommon for 15 to 20 per cent of the plants to be diseased.

The Ascomycetes

This is a large and varied group. The type character which marks it from the other groups is the spore bearing body (ascus) which resembles a sack and from which the class takes its name.

The asexual reproduction in the ascomycetes consists of the mycelium, the conidiophorese, conidiae and, in some cases, of sclerotial masses which function as resting bodies. Typical examples of the asexual reproduction are illustrated in Plate No. II, E, 1 and F,

The conidia which are usually borne in chains are oblong to round in shape, thin-walled and nearly colourless. They are short-lived but as they are produced in enormous numbers they are a vital factor in the distribution of the disease during the active growing period of the host plant. They are scattered by wind, rain, insects and other agents.

The sexual reproduction of the *Ascomycetes* is a somewhat complicated process. Antheridia and oogonia are developed on semi-sclerotial masses of mycelium. On Plate No. II, H, are some of the steps in diagram. The oogonium and the antheridium are uninucleate. Conjugation takes place between them and the fertilized oogonial cell then divides to give rise to six nuclei. Cell walls are laid down between nuclei 1 and 2, 2 and 3, 3

PLATE II

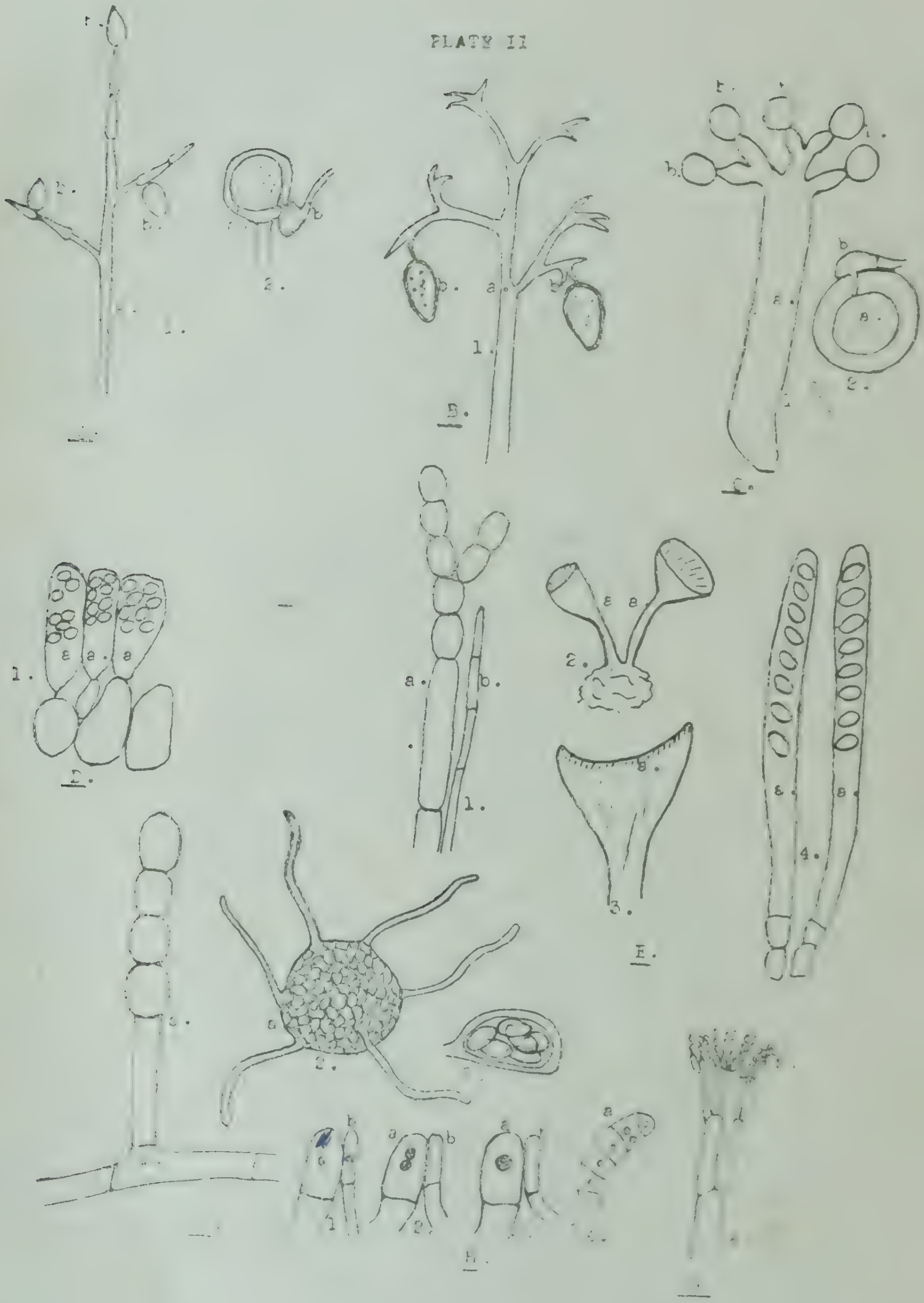

Plate No. II.

A. *Phytophthora.*
  1. Asexual reproduction. a. Conidiophore. b. Conidia.
  2. Sexual reproduction. a. Oogonium. b. Antheridium.

B. *Perpnospora.*
  1. Asexual reproduction. a. Conidiophore. b. Antheridium.

C. *Sclerospora.*
  1. Asexual reproduction. a. Conidiophore. b. Conidia.
  2. Sexual reproduction. a. Oogonium. b. Antheridium.

D. *Taphrina.*
  1. Asexual reproduction. a. Asci and ascospores.

E. *Sclerotinia.*
  1. Asexual reproduction. a. Conidiophore with conidia. b. Paraphysis.
  2. Sexual reproduction. a. Apothecium. Cupshaped body containing asci.
  3. Longitudinal section of an apothecium.
  4. Asci (a) and ascospores.

F. Powdery Mildew. *Erysiphe polygoni.*
  1. Asexual reproduction. a. Conidiophore and Conidia.
  2. Cleistothecium containing asci.
  3. Ascus with ascospores.

G. *Penicillium.*
  1. Diagram of asexual stage. a. Conidiophore and b. Conidia.

H. Phases in the sexual reproduction of an *Ascomycete.*
  1. a. Oogonium containing a single nucleus. b. Antheridium containing a single nucleus.
  2. a. Oogonium with two nuclei. One from the antheridium and one its own. b. Empty antheridium.
  3. a. Oogonium in which the two nuclei have united. b. Empty antheridium.
  4. The column of cells developed from the fertilized oogonium. a. The two nucleated, penultimate cell from which the ascus develops.

and 4, and 5 and 6, but not between 4 and 5. This binucleated cell is often referred to as the penultimate cell. These two nuclei fuse and then from the fusion there are formed, by division, typically 8 nuclei. The cell enlarges and each nucleus, together with a portion of the cytoplasm, is surrounded by a cell wall and becomes an ascospore. The whole structure becomes an ascus.

The asci vary according to the species, the ascus type being one of the characters upon which the various groups have been set up. Plate No. III, D, 1, 2, 3 and 4 are illustrations of types of asci. Other types are illustrated in Plate No. II, D, 1 and F, 3.

The ascocarp, the body that bears the ascus, is also used as a base for the classification. The ascocarp varies from one that is completely closed (a cleistothecium) Plate No. II F, 2, as in the powdery mildews, to one that is flask-shaped with an opening, ostiole (a perithecium), Plate No. III C; to forms that have a disc-shaped fruiting structure, as illustrated in Plate No. II F. In some there is no fruiting structure at all and the asci are borne on the surface of the leaf, as illustrated in Plate II D. There is great variation among the flask-shaped ascocarps. Some are short-necked and are produced on the surface of the host tissue, whereas others have a long neck and are deeply imbedded beneath the host tissue.

The ascospores also vary. Some are unicellular, as shown in Plate No. II D, E and F. Some are bicellular, as illustrated in Plate No. III D, 2. Some are multicellular and even filamentous, as shown in Plate III D, 3 and 4. The ascospores are short-lived as a rule but as they are produced only when the host plant is in a receptive stage they are usually effective in spreading the disease. The spores are often forcibly discharged from the ascus and then carried by the wind to the host plants. This discharge is brought about by the absorption of moisture under the heat of the sun and, when the increasing moisture becomes too great for the ascus, it ruptures with the forcible discharge of the spores. While wind is an important agent, at the same time water, insects, man and other agents are also important as carriers.

Many of the most serious plant diseases are found among the *Ascomycetes.* Examples would be the powdery mildews, especially of the genus *Erysiphe;* bean anthracnose, *Colletotrichum lindemuthianum,* the perfect stage of which is *Glomerella cingulata;* cotton anthracnose, *Colletotrichum gossypii,* the perfect stage of which is *Glomerella gossypii;* the die-back of *Citrus, Colletotrichum gloeosporioides,* the perfect stage of which is *Glomerella cingulata; Nectria cinnabarina,* the canker on tea; brown rot of stone fruits *Sclerotinia fruticola; Taphrina deformans,* peach leaf curl and many others.

The Basidiomycetes

This is a large and variable group and contains many of the very serious plant pathogens. The type character of the group is the basidium. This is the mycelium produced when the resting spore germinates. The basidium varies with the different members of the class and is one of the major characters upon which the division of the class into sub-class and orders has been based. The class is divided into three main groups commonly referred to as the smuts, the rusts and the toad stools and mushrooms. The smuts derive the name from the fact that the portion of the host infected becomes filled with a mass of brown or black spores (chlamydospores). In some cases, as for example, the ear smut of maize and the kernel smut of many cereals, the entire plant part becomes transformed into a spore mass containing millions of spores. (See under the section on Plant Disease through the Air).

The chlamydospores of the common smuts are formed as the result of the laying down of cross walls

PLATE III

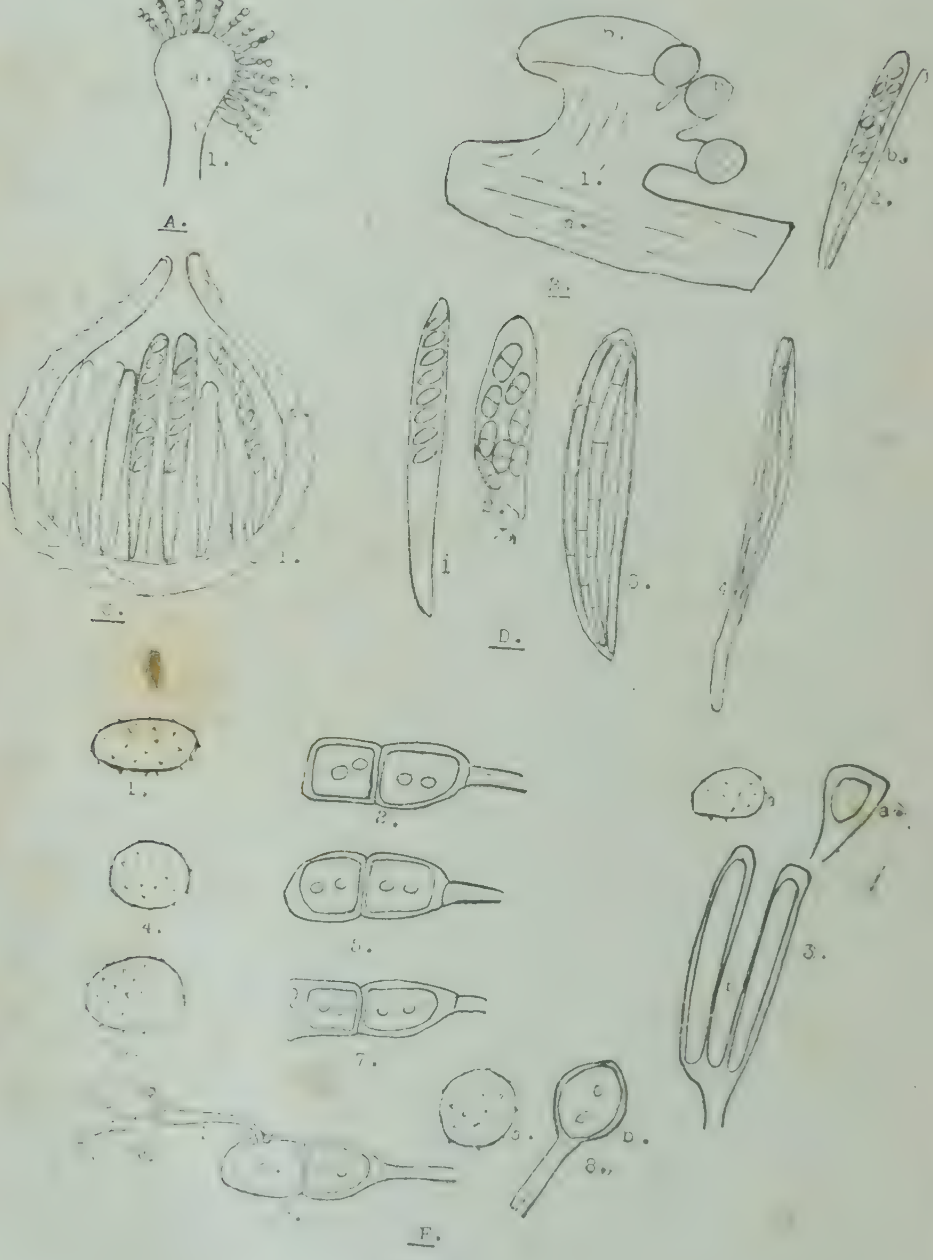

Plate No. III.

A. *Aspergillus.* a. Conidiophore. b. Conidia.

B. *Nectria.*

1. Longitudinal section of a canker. a. Host tissue. b. Sclerotial mass. c. Perithecia containing asci and paraphyses.
2. Single ascus and a paraphysis. a. ascus and ascospores. b. Single paraphysis.

C. Typical perithecium of an *Ascomycete* such as *Mycosphaerella.*

1. a. The perithecium wall. b. Asci. c. Paraphyses.

D. Typical types of asci.

1. Ascus with single celled asci. Example *Pseudopezizza.*
2. Ascus with two celled ascospores. Exampde, Apple scab, *Venturia inequalis.*
3. Ascus with multiseptate ascospores. Example *Ophiobolus.*
4. Ascus with filamentuous ascospores. Example *Ergot.*

E. Examples of rust spores.

1. Uredospore of *Puccinia graminis.*
2. Teleutospore of *Puccinia triticinia.*
3. Linseed rust. *Melampsori lini.* a. Uredospores. b. Teleutospores.
4. Uredospore of *Puccinia triticina.*
5. Teleutospore of *Puccinia graminis.*
6. Uredospore of *Puccinia glumarum.*
7. Teleutospore of *Puccinia coronata.* (Crown rust of oats).
8. *Uromyces striatus.* a. Uredospore. b. Teleutospore.
9. Germinating teleutospore. a. Germinated cell. b. basidium. c. Basidiospores.

in a multinuclear mycelium. In most cases conjugation occurs between the nuclei of adjacent cells and, as a result, each chlamydospore is binucleate. The nuclei fuse just before germination and then reduction division takes place just before the formation of the basidiospores. The method of germination and the formation of the basidiospores is also a character used in the division of the order into families. The *Ustilaginaceae*, in which are found the loose smuts, produces a septate basidium and the basidiospores are produced along the side. The *Tilletiaceae*, in which are found the bunts and covered smuts, produce a non-septate mycelium and the basidiospores (basidia) are produced at the end in a cluster. The basidiospores are of two types, i.e., male or female. Fusion between cells of opposite sex is the rule before infection of a host plant can be successful. In some cases this occurs after the penetration of the host tissue by the promycelium of the germinating basidiospores. For example, in the case of the smut of maize, the fungus may penetrate the host plant but there must be present the mycelium of both types of basidiospores and these must fuse before any further invasion can take place or any spore mass form. In the case of the bunts, fusion of the sporidia may occur while they are still on the basidium and thus the invading mycelium is self-sufficient for the production of the spore mass.

In most of the smuts the chlamydospores carry them over the adverse part of the season. The spores may remain in the old dead plant tissue, as in the case of leaf smut of maize; they may be within the seed, as in the loose smut of wheat; they may remain on the outside of the seeds, as in the case of covered smut of barley, oat smut; or they may remain in the soil, as in the case of maize smut. Life cycles and the various methods of infection are given under the specific heading in the chapters on plant diseases.

Among some of the more important of the plant diseases caused by smuts can be listed the smut of maize, the loose smut of jowar, wheat, barley and oats, the kernel smuts of bajra and of jowar, and the covered smuts of wheat and barley. Some of the more spectacular smuts are the long smut of jowar and the sugar cane smut.

The rust fungi

These are probably the oldest known of the plant diseases. Ancient literature records blights and blasts of the crops in the Nile Valley and in the central portions of Asia and along the trade routes. It is not always clear what grain is referred to but it is generally assumed that wheat or barley is referred to and that stem rust is the disease. Then it was held that the gods were angry and the people had sinned. Without microscopes and unable to see the minute organisms, the people were ignorant of the real cause until about the middle of the last century (1865) when Anton de Bary published the first account of a rust life cycle. At that time he reported on the life cycle of black stem rust of wheat in Germany. This report opened the way for the study of other rusts and soon the cause of the scourge of the wheat fields was better understood.

A study of the rusts has led to the general belief that the species consist of two states of development. These two states are the gametophyte and sporophyte stages. The gametophyte stage, the haploid stage, is relatively short. The sporophyte stage is the diploid stage and is longer. During the gametophyte stage the pycnospores (sporidia) and aecidiospores are formed. (See under *Basidiomycetes* in Chapter II and under black stem rust of wheat in Chapter VI). During the sporophyte stage the uredospores and teleutospores are formed. These two stages may

PLATE IV.

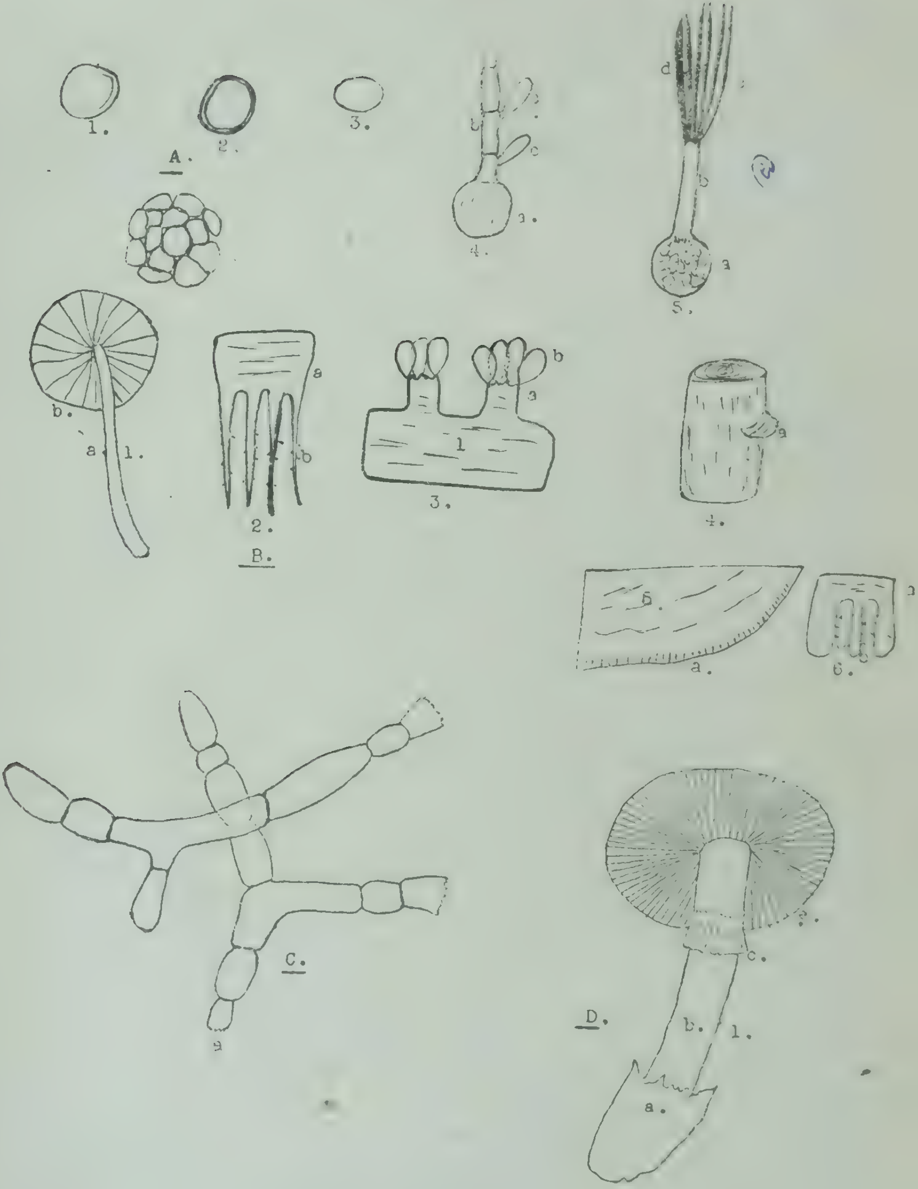

Plate No. IV

A. Examples of smut spores.
  1. Spore of bunt.
  2. Spore of loose smut of barley.
  3. Spore of loose smut of oat.
  4. Germination of loose smut. a. Chlamydospore. b. Basidium. c. Basidiospores.
  5. Germination of spore of bunt. a. Chlamydospore. b. Basidium. c. Basidiospores. d. Basidiospores with conjugation tube.
  6. Spore ball of *Urocystis.*

B. Examples of the true or higher *Basidiomycetes.*
  1. *Agaricus.* a. Stipe. b. Pileus.
  2. Diagram of a section of the pileus a. and gills b.
  3. Diagram of section of pileus showing spore production. a. Basidium. b. Basidiospores.
  4. Diagram of portion of tree trunk with a sporophore of a polypore. a.
  5. Diagram of a portion of a sporophore showing position of the pores a.
  6. Diagram showing the pores. a. Sporophore. b. Pores with spores. Note. Basidia and basidiospores very similar to those of *Agaricus.*

C. Example of mycelium of *Rhizoctonia* one of the *Mycelia* sterilia. a. Basidium.

D. Diagram illustrating the poisonous type of mushroom.
  1. a. the vulva. b. Stipe. c. Annulus. d. Pileus.

be on the same host plant, in which case, the rust is said to be "autecious." They may be on separate host plants in which case they are said to be "*heteroecious*." In some of the rusts there are two kinds of spores as regards, what appears to be, sex. One of these seems to function as the male and the other as the female. (See under black stem rust of wheat). Where this condition occurs the rust is said to be "*heterothallic*."

Many of the rusts do not form all of the spores. That is some of them are omitted. Modifications have taken place and some of the spores appear to have been lost. The most common and best known genus of rusts is the genus *Puccinia*, which contains most of the cereal rusts. (See under Chapter II) In general the genus contains all of the spore forms but there are some rusts that do not contain all of the spore forms and there may be a slight difference between some of the spores, otherwise they appear identical. These forms are placed in other genera. An example of this sort would be that of the genus *Uromyces*, which is almost identical with *Puccinia*, except for the single-celled teleutospores. Some of the rusts produce no uredospores, others may produce only teleutospores, some produce no known teleutospore forms and so it goes.

The rusts receive their names from the appearance on the host plant. The most common character for field identification is the red, yellow, brown or black sori over the leaves and stems which, when seen in large numbers, give the plant the rusty appearance. They are extremely widespread and occur on practically every known farm crop. Arthur (31) lists some 694 species of rusts of which over half have been recorded in India. A number of rusts recorded in India are not recorded by Arthur and *vice versa*. The losses each year due to the rust fungi are large. In 1947 wheat losses in several parts of India had a very direct effect upon the wheat supply. Gram suffered from rust in the Punjab and Central India. Peas and alfalfa suffered from rusts in parts of the United Provinces.

The severity of the rust attack is closely correlated with the weather. (See under Chapter V) A high humidity and moderate temperature being the optimum for the fungi. There are some eight or ten forms of the black stem rust of wheat in India. Forms 15, 21, 24, 34, 40, 42, 75, A & B are quite definitely established. Of these forms 15, 21, 40 and 42 are known to be in the United Provinces with form 42 being the most widely distributed. Eight forms of leaf rust of wheat have been indentified as follows; forms 10, 20, 26, 63, 106, 107, 108, and D. of these forms 10, 20, 63 and 107 are in the United Provinces.

Among the fungi, called the true fungi, are many which produce very large fruiting bodies. The more common ones known to most of us are the toadstools and mushrooms. Although there is no hard and fast rule, the general practice has been to call the gill fungi the mushrooms and the pore fungi the toadstools. The gill fungi (see Plate No. IV, B) are those which have small curtain-like structures hanging from the under portion of the pileus. They are classed under the order *Agaricales*. The toadstools, on the other hand, are those fungi which have the under surface of the pileus pitted with small pores. These fungi are referred to as the polypores and are classed under the order *Polyporales*. The spore-bearing layer, hymenium, is found on each side of the gills in the *Agaricales* and lining the pores of the *Polyporales*. There are numerous other groups of the true fungi, each different in the form and structure of the fruiting body. Some of them are important as wood rotting fungi and some are serious on the roots of some of the important crop plants of farm and forest. Butler and Bisby (96) list some eighty species of the genus *Polyporus* which have been found in India, mostly on dead wood. They list eleven species of *Corticium*, including *C. invisum*, the cause of black rot of tea; *C. koleroga*, which has been reported on the leaves and twigs of coffee; *C. repens*, the cause of thread blight of tea; *C. salmonicolor*, which has been reported on the living wood of *Hevea*, *Thea*, *Coffee*, *Cinchona* and *Citrus*. They report some thirty-six species of *Fomes*, which includes a number of the important forest-tree-rotting fungi.

Among the mushrooms and toadstools are a number which are extremely poisonous. It is a common adage that, for the amateur, it is better to leave the white spored agarics alone. Among the *Agaricales* possessing white spores are the *Amanitas*. These are recognizable by the white gills and spores, the ring about the stipe just beneath the pileus and the cup (Vulva) at the base of the stipe. (See Plate No. IV, D).

The Imperfect Fungi

The imperfect fungi are so listed because the perfect, or sexual, stage, is unknown. The only reproductive stage known is the vegetative. On the basis of the known vegetative phases the class has been divided into three main groups. In one group are placed those forms which possess a fruiting body shaped like a flask and with an opening, or ostiole, through which the spores escape. The mycelium of this group is not extensive but there is a stromatic mass, formed by many of the species, which produces the flask-shaped fruiting bodies or pycnidia (singular, pycnidium). The spores formed in these bodies are known as pycnospores and they vary from small, nearly round, unicellular forms to those that are more or less filamentous, multicellular and many times the length of the round types. In fact, pycnospores are really specialized types of conidia which are produced in a specialized fruiting body.

The pycnospores are usually short-lived but are produced in enormous numbers and thus they are a major factor in the spread of these fungi. These fungi do not depend upon the pycnospores to carry them over the adverse season but for the most part it is the stromatic mass that remains alive and forms the first spores produced when the normal growing season of the plant reappears. Many of the most destructive of the plant diseases are caused by fungi of this group. Among the more common diseases are the *Phyllosticta* leaf spots of farm crops. There is one on the papaya, one on the guava, a canker on brinjal stems, a leaf spot on roselle (patwa), one on pigeon pea. The pycnospores of *Phyllosticta* are small and nearly round. The genus *Septoria,* possesses long, slender, multicellular spores. Butler and Bisby (96) list some thirty-nine species of *Septoria* which have been reported on plants in India. Several of these are of importance, including one on wheat, one on soya-bean and one on the hyacinth bean (*Dolichos lablab*).

Another group of the *Imperfecti* is based on the conidia being borne on a saucer-shaped fruiting body, the acervulus. While not a large group, some of the most destructive plant diseases are found among them. Such diseases as the red rot of sugarcane, the red spot of jowar, the die back of *Citrus,* the anthracnose of beans and of papaya are examples. Many of the most important of these fungi belong to the genus *Colletotrichum.* A number of these have been studied and the life cycle completed so that the perfect stage is known. (See the section under *Ascomycetes*).

The third group of the *Fungi Imperfecti* contains those which have no specialized fruiting body and which produce the spores on the open mycelial mass. In this group are a wide range of types. The spores vary from single-celled, small, almost spherical, to those that are large, multicellular and which may be filamentous, muriform, straight or curved. As in the case of the previous groups, some of these fungi produce some very destructive diseases. The *Cercospora* leaf spots are found on nearly all the common farm crops. There is

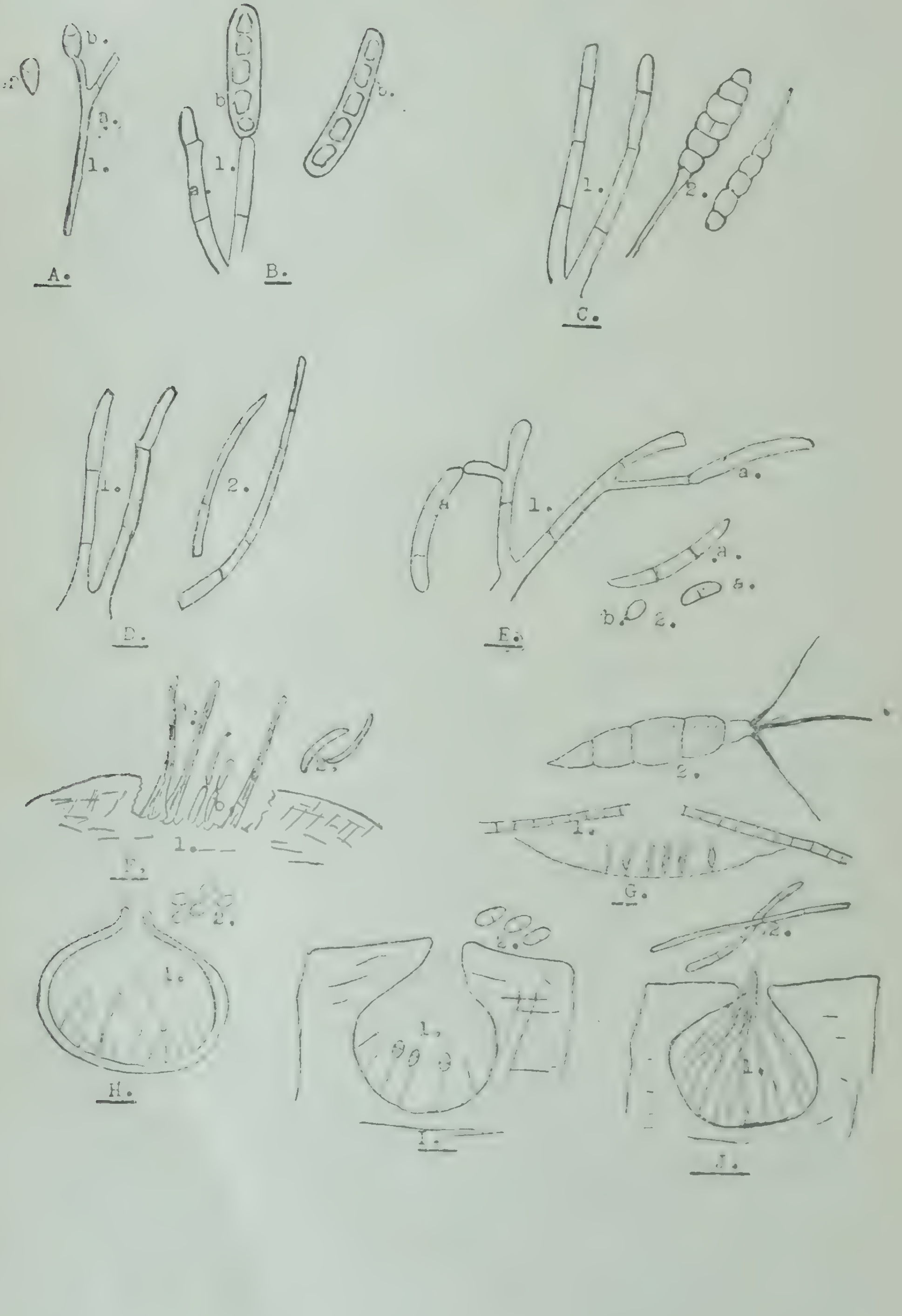
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Plate No. V

A. *Piricularia.*
    1. a. conidiophore and b. conidium.
    2. Single conidium.

B. *Helminthosporium.*
    1. Conidiophores and conidia. a. Conidiophore. b. Conidia.

C. *Alternaria.*
    1. Conidiophores.
    2. Conidia.

D. *Cercospora.*
    1. Conidiophores.
    2. Conidia.

E. *Fusarium.*
    1. Conidiophores and a. Conidia.
    2. a. Macroconidia. b. Microconidia.

F. *Colletotrichum.*
    1. Diagram of an acervulus. a. Setae. b. Conidiophores and conidia.
    2. Conidia.

G. *Pestalotsia.*
    1. Diagram of an acervulus.
    2. Single conidium.

H. Phoma type pycnidium.
    1. Diagram of a pycnidium containing pycnospores.
    2. Pycnospores.

I. *Diplodia* type.
    1. Diagram of a pycnidium containing pycnospores.
    2. Pycnospores.

J. *Septoria* type.
    1. Diagram of a pycnidium and pycnospores.
    2. Pycnospores.

one on potato, one on the groundnut, one on maize, one on bajra, on brinjal, tobacco and many other farm crops. The *Alternaria* leaf spots are found on many farm crops. Early blight of potato is well known. There is one on cabbage, on radish, on linseed and one on tomato. The *Helminthosporium* leaf spots are common on many farm crops. Maize, jowar, wheat, barley and many other cereals are attacked. The *Fusarium* rots and wilts are common everywhere. They offer a particularly serious problem because they are often soil borne and thus attack the roots of the host plant.

The question of recognizing the diseases in the field is important. In many cases the symptoms are very distinct, while in others they are very confused. The pycnidia-forming fungi are not so difficult if one has a hand lens. The pycnidia are the distinctive character. In general it can be said that a pycnidia-forming fungus with small, spherical, single-celled spores, when occurring on leaves, belongs to the genus *Phyllosticta.* If the same type of fruiting body and spores occur on a stem it is of the genus *Phoma.* Classifying fungi according to the plant parts affected is exceedingly artificial and one that is not likely to stand for a very long time. At this time, however, a pycnidia-forming fungus infecting the leaves of crop plants is considered a *Phyllosticta* and one on the stem, a *Phoma.*

Those fungi which form acervuli often produce more or less round infection spots, which, if on fruits or stems, are sunken in the centre. The common term anthracnose has been applied to many of these, such as the anthracnose of beans, melons and papaya. Under favourable conditions for the formation of spores, they are produced in large masses and in many cases assume a yellowish, orange or pinkish colour in mass. This is especially true of the three diseases just mentioned. Acervuli are not usually clearly discernible with the unaided eye. A character that is of aid in the quick identification of some of the anthracnose disease fungi is the presence of setae. These are stiff, brown or black, hair-like structures that arise around the margin of the acervulus. Occasionally they may be visible to the naked eye, but more often require a hand lens to see and in some cases they are not present. Perhaps the most common example of an acervulus-forming fungus is the one causing red spot of jowar. On the older infected areas the acervuli may be seen in large numbers.

The *Cercospora, Helminthosporium, Alternaria* types of fungi are identified by the characters of the infected area. Hand lens are of value but it may be that there are no spores being formed on the mycelium and thus one has to rely on the microscopic characters. The spots formed by each of the three genera mentioned are more likely to be found on the leaves. *Cercospora* leaf spots are likely to be rounded in shape, from one to several mm. in diameter and with a grayish centre. The margin may or may not be dark. That will depend more upon the age of the spots and the host plant. *Helminthosporium* spots are likely to be much more elongated, grayish-margined and darker centres and in some cases the diseased area will split into shreds. This is especially true of such diseases as barley stripe. The *Alternaria* spots will resemble the *Cercospora* spots but are likely to be darker and with concentric rings marking the central portion of the area. That is the easiest way of differentiating between *Cercospora* and *Alternaria* on *potatoes.* The other leaf spot diseases need to be studied in the field to make identification at all certain. Even then it is often necessary to take the leaves to the laboratory where it is possible to make use of the compound microscope or to make isolations and thus study the fungus on artificial media.

Mycelia Sterilia. This is a small group of fungi, considered for a number of years to be sterile and thus given the above name. The most common fungi of this group are *Rhizoctonia* and *Sclerotium.*

Some of the species of *Rhizoctonia* have been found to produce basidiospores and thus to be *Basidiomycetes* but the majority are still considered as sterile. The resting body is called a sclerotium and the easiest means of identification is the sclerotium, where found. *Sclerotium rolfsii,* common on a large number of crops, such as groundnut, potato, barley, etc., produce small round sclerotia that resemble mustard seeds in size and, somewhat, in colour. The sclerotia of *Rhizoctonia* are irregular in shape and when found on potato tubers are often referred to as "the dirt that will not wash off". As they are living fungus bodies, they grow close to the tuber skin and if pulled off they will usually take a portion of the skin with them. The most common examples of the *Mycelia sterilia* are likely to be *Rhizoctonia solani* and R. *bataticola,* found on potatoes and many other farm crops, *Sclerotium rolfsii* and S. *oryzae,* the former already referred to and S. *oryzae* common on rice.

The Virus Diseases. These are ultra microscopic pathogens that cannot be seen with the ordinary microscope. In fact it is not yet certain that they are visible even with the new and extremely powerful microscopes. While plant physiologists and chemists have been attempting to visualize the structure of an organic compound, or organism, that will answer to the known characteristics of the viruses, as yet it does not appear that any one is positive as to just what they are like. Any descriptions which may be given of a virus disease are in terms of the appearance of the host plant and not of the pathogen.

Virus diseases are difficult to differentiate in most host plants. This is especially true of those hosts on which a number of virus diseases appear at the same time so that the characters may be confused. The outstanding characteristic of the viruses is to cause changes in the chlorophyll patterns. Since changes in the chlorophyll can also be caused by light, water or minerals, it is often puzzling to know how much of a change in colour is due to the virus, or viruses, and how much due to other factors. The colour changes in the chlorophyll pattern vary from variation in colour that occur in irregular-shaped patches to those that involve the entire leaf. In some cases they are in streaks, as in the case of sugarcane mosaic.

In some cases the effect of the virus is to cause malformations in the stems and leaves. Stunting, spindling of the stem, reduction in size of leaves, sterility of the flowers are common characters associated with virus diseases.

The number of virus diseases recognized is enormous. (See section under Classification). In many cases it is doubtful if the number is really justified. Many of the diseases, based on symptoms, are really the same virus but producing different symptoms under different conditions.

In the field the major virus disease symptoms one sees are the irregular patterns in the chlorophyll, the stunting and deforming of the tops, the lack of fruitfulness and dying of plants without external evidence of necrosis.

Transmission of the viruses is largely by insects but there are also many other agents which are capable of carrying the pathogen from one plant to another. Birds, animals, men, tools and, more recently it has been shown (Johnson 318) that such parasitic plants as doddar may also transmit the disease from one plant to another.

CHAPTER II

THE CLASSIFICATION OF FUNGI

There is no phase of botany or zoology requiring more patient careful observation than that of taxonomy or the classification of living things. Because of the exacting requirements, few ever become really outstanding taxonomists. That does not mean that the amateur has not made any contribution to the field of biological classification for that is very far from the truth. A vast amount of the present mass of classification data has been collected by amateurs. But the outstanding contributions to the field have been made by a comparatively few. The whole system is complex and requires a special ability so that the general trends in the direction of the classification must be in the hands of the experts. Work of amateurs must be examined by competent authorities before being adopted for general use.

The one who classifies fungi is called a mycologist and the field in which he works is known as mycology. Mycology is the oldest of the phases of fungi study. Applied mycology and its closely related phase, plant pathology, are much more recent. In the very beginning men classified fungi much as they did all other plants, merely giving them common names which may have related to the place they were first found the men who first found them, or some other relationship which struck the fancy of the collector. As a result the same fungus might be given a different name in each section of the country. This led to confusion. In the 16th century there was some attempt to bring order into the field on the part of the German doctors along the Rhine Valley. But this effort was without leadership and direction.

There were no periodicals at that time which devoted any space to plant classification. Communication was limited and as a result there was little opportunity for men to get together to set up any general system.

In the 17th century the first step was made toward the solution of the problem. From 1835 to 1851 Carl von Linnaeus established the binomial system of nomenclature which has not been improved upon. It has been the foundation of the present system of taxonomy. "Binomial" means two names. The first name is the generic name and the second is the specific name. The two together make up the scientific name of an animal or plant. Thus the common black bread mould is known as *Rhizopus nigricans* Ehr. *Rhizopus* is the generic name and *nigricans* is the specific name. With this system as a basis it was possible to classify all of the living things so that they could be put in an orderly arrangement. This was impossible before. It is true that much of the work done by Linnaeus has not been practical but the setting up of the binomial system for others to follow was one of the really great landmarks in the history of botany. It provided the framework for taxonomy.

The providing of a system of classification is one thing but the arrangement of the many different species of living things in the proper genera, families, orders, classes, etc., is a tremendous task. It requires that all observations must be accurate and complete. Descriptions must be carefully done. Drawings exact, clear and distinct. The important structures must be shown.

For the beginner two little books have recently appeared that will be of great aid. "An Introduction to the Taxonomy and Nomenclature of Fungi" by G. R. Bisby and "Outline of the Fungi" by G. W. Martin are extremely worthwhile. Martin (421) has simplified the classification as to the number of characters used making it easier to follow. This work is being followed largely in this outline.

THE PLANT KINGDOM

The plant kingdom has been artificially divided into four subdivisions. The *Thallophyta*, the *Bryophyta*, the *Pteridophyta* and *the Spermatophyta*. The order of rank being in degree of development from the most primitive to the most highly developed. The *Thallophyta* being the lowest, or the most primitive. These in turn are subdivided into two subdivisions on the basis of the metabolism.

I. ALGAE—Those *Thallophytes* that possess chlorophyll.

There are a few cases in which algae cause plant disease, as in the case of the red algae (*Cephaleuros virescens*) which may be found growing on the green portions of the mango, but they usually cause little more than an interference with the passing of the sunlight.

II. FUNGI—Those *Thallophytes* which do not have chlorophyll.

The fungi are subdivided into classes based upon the morphology of the fungus body or upon the reproductive phases. Some mycologists are inclined to exclude the bacteria from the classification of the fungi, placing them between the *Algae* and the *Fungi* as a separate group. But, as the majority of the workers at this time still retain them among the *Fungi*, they will be presented at this time.

A. *Schizomycetes*

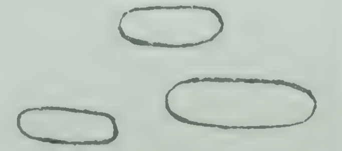
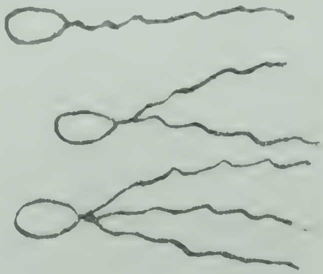
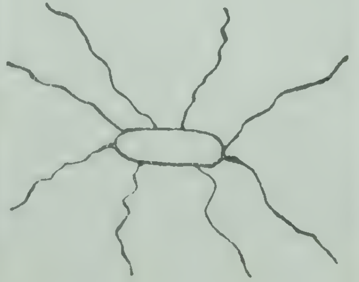
These are single-celled organisms which reproduce by fission or schism (a rent). It is because of the method of reproduction that they are given the name *Schizomycetes*. They may be single, in pairs, in chains or in the form of squares or cubes. The nucleus may be lacking or very poorly defined.

Bacteria were not associated with plant disease until about 1880 when Professor Burrill, of the University of Illinois (U. S. A.), demonstrated that fire blight of apples was caused by a species of *Bacterium*. Following this discovery there was a definite interest directed along the lines of plant bacterial disease research. In 1896, Erwin F. Smith of the United States Department of Agriculture, predicted that there were, in all probabilities, as many bacterial plant pathogens as there were animal bacterial pathogens. In 1920 he reported the occurrence of specific bacterial plant diseases on plant hosts scattered through more than 150 genera and over 60 families. In 1931 Charlotte Elliott (207) listed the bacterial plant pathogens under seven genera as follows: *Aplanobacter* 13 species; *Bacillus* 60 species; *Bacterium* 111 species; *Clostridium* 1 species; *Pseudomonas* 1 species; *Phytomonas* 1 species; *Micrococcus* 1 species, The number of species has been constantly enlarging and today is much larger than when Miss Elliott wrote her book, (Manual of Bacterial Plant Pathogens).

The more important symptoms produced by bacteria are soft rot, wilting, necrosis, watersoaking of the tissues and chlorosis. Bacteria are disseminated in various ways. The more important ways of dissemination are: by seed, by parts of the plant used in propagation (potato, sugarcane, etc.) by insects and birds, by rain, by wind, by man, animals and tools used in cultivation.

The classification of bacteria has been as badly confused as that of the other sections of the plant kingdom. The bacterial plant pathogens have been placed under some three genera. These have not always been given the same names but the number has remained approximately the same.

The following tabular arrangement from Heald (275) of the types of bacteria will help make clear the differences between the three systems as regards the plant pathogens.

Non-motile	Motile. Flagella polar	Motile. Flagella peritrichous	Author
			
Bacterium	*Pseudomonas*	*Bacillus*	Migula
Aplanobacter	*Bacterium*	*Bacillus*	E. F. Smith
Phytomonas	*Phytomonas*	*Erwinia*	Society of American Bacteriologists.

In 1939 Dawson (175) after studying some 93 carefully authenticated organisms which were Gram negative and 7 that were Gram positive, concluded that they should be grouped into three groups according to their behaviour on specific media. Accordingly he set up the following three groups:

1. *B. coli-like.* Those organisms which form acid and gas in salicin.

2. *P. flourescens-like.* Those which do not produce acid and gas, in lactose, maltose or salicin.

3. Those which produce, on solid media, a characteristic, abundant, slimy yellow growth but no acid in salicin.

His contention was that a genus cannot be set up on one character alone, as for example, the case of *Bacterium* and *Aplanobacter,* on non-motility or pathogenicity; or as in the case of *Erwinia* and *Phytomonas,* on motility. For that reason he says that *Bacterium* should be applied to group 1 and *Pseudomonas* to group 2. In 1937, Elliott (207) showed that the plant genus *Phytomonas* was antedated by a flagellated protozoan genus *Phytomonas* of 1909 and therefore is untenable. To the group 3 Dawson gave the name *Xanthomonas.* The characteristics of the three groups would be as follows:

Bacterium. Acid and gas formed in salicin. Non-spore forming, rod-shaped, Gram negative, motile by means of peritrichous flagella, or non-motile, gray or transparent on nutrient agar, forming creamy, later yellowish, growths, on potato.

Pseudomonas. Acid or gas not produced in lactose, maltose and salicin; non-sporing, rod-shaped, Gram negative, motile by means of polar flagella (1-7 in number, never non-motile), white or transparent on beef infusion and on starch agar, on both of which fluorescin is produced by most species, forming creamy, later pink, growths on potato.

Xanthomonas. Non-sporing, rod-shaped, Gram negative, uni-, rarely bi-flagellate or non-motile, forming abundant yellow, slimy colonies on nutrient agar and potato, mostly digesting starch and producing acid in lactose but not in salicin. In this group are placed a large number of species which have been hitherto found under the genera, which are being abandoned in making up this classification.

The work of Dawson has been largely accepted by the plant pathologists and it will be followed in this outline. A complete classification for any species may be represented by the following sample—

Division—*Thallophyta.*
Class—*Schizomycetes.*
Order—*Eubacteriales.*
Family—*Bacteriaceae.*
Genus—*Bacterium.*
Species—*Bacterium coli.*

B. *Myxomycetes*

This class of fungi is often claimed by both zoologists and botanists. It possesses a body structure that is one time amoeboid in nature and, at another time, quite plant-like. The class is further subdivided into subclasses on the basis of the manner of spore production.

1. *Exosporeae.* Spores borne externally.
2. *Myxogastre.* Spores borne internally.

There are few slime moulds that are serious as plant pathogens. In the temperate regions club root of cabbage is a serious disease which is caused by one of the slime mouds, *Plasmodiophora brassicae* Woronin.

C. *Phycomcetes*

These are the algae-like fungi. They are so named because of their resemblance to some of the algae. In the past they have been referred to as the *Siphonomycetes* because of the resemblance to some of the green algae but this name has not become widely used in literature. It has been suggested that they may be degenerate algae. The chief characteristic between this class and the next higher ones lies in the coenocytic (without cross-walls) mycelium. The only cross-walls usually found present are at the point where the sex organs are formed. The *Phycomycetes* may be further subdivided into three subclasses as follows—

1. *Archimycetes*

These are the most primitive of the *Phycomycetes.* They are naked, often amoeboid in form and the reproductive organs are developed by a division of the whole thallus. In this character they show relationships with the Myxomycetes. The vegetative body is one-celled and may be naked or possess a covering membrane. Reproduction, both sexual and asexual, consists of zoospores and sporangia. Sexual reproduction consists of a copulation between specialized cells. Two orders are usually recognized.

a. *Plasmodiophorales*

The thallus is a naked body at maturity with a spore mass that may be naked or with a membrane covering it. The spores germinate to produce swarm cells motile with two unequal anterior flagella. These orders include a number of parasites on vascular plants causing hypertrophy. There is one family—*Plasmodiophoraceae. Plasmodiophora brassicae* Wor., club root of cabbage being an example.

b. *Myxochytridiales*

The members of this sub-class always have a membrane covering the spore mass. The spores germinate to produce swarm cells but never with unequal flagella. Three families are set up under the order. *Woroniaceae*, *Olpidiaceae* and *Synchytriaceae.*

2. *Oomcetes*

The first well-developed mycelium is found in this class. The hyphae are capable of penetrating the substratum upon which the fungi live and thus enabling them to be either saprophytes or parasites. The gametangia are unlike. The sexual stage is represented by oospores; the asexual stage by zoospores or sporangia that eventually produce zoospores. In some cases the sporangia germinate directly.

The *Oomycetes* are sub-divided into five orders on the basis of the reproductive and vegetative phases.

a. *Monochytridiales* (*Chytridiales*)

These forms possess a sterile thallus portion which is only a basal haustorium or by a very scanty mycelium. Mostly parasitic on water and land plants. The order contains two families as follows: the *Rhizidiaceae* and the *Cladochytriaceae*. In the family *Cladochytriaceae* are the genera *Cladochytrium*, *Physoderma* and *Urophlyctis*. The latter two genera are represented by plant disease fungi in India.

b. *Lagenidiales*

There is a considerable mycelial growth among the members of this order. However the growth is often confined to a single cell. At maturity it may be transformed entirely into reproductive structures. A single family, *Lagenidiaceae*.

c. *Blastocladiales*

In this order there is abundant mycelium which never completely transforms into reproductive structures. The gametes are heterogametes with egg and sperm. The zoospores are uniflagellate. Two families are found under this order.

(1) *Blastocladiaceae*. Gametes morphologically similar.

(2) *Monoblepharidaceae*. Gametes dissimilar.

d. *Saprolengniales*

Oospores usually several to many. Zoospores attached to mycelium. Mostly saprophytic but few parasitic. There are two families based on the oospore formation.

(1) *Saprolegniaceae*. Usually several oospores. No constriction of the hyphae at the point of formation of the oospore.

(2) *Leptomitaceae*. Only one oospore but with a constriction of the hypae at fairly regular intervals.

e. *Peronosporales*

These are characterized by a richly-branched mycelium, which is thread-like, much-branched, typically one-celled (coenocytic) until the reproductive organs are formed and then septate only at the point of formation of the gametes. Sexual reproduction by heterogametes. The male ~~gamete~~ [illegible] (antheridium) is a club-shaped hypha, usually arising near a female reproductive organ (oogonium). The oogonium is a large spherical organ, at first thin-walled and may be either uninucleate or multinucleate. The antheridium is usually uninucleate. If the oogonium is multinucleate only one will function as the egg nucleus. The other nuclei degenerating into the peripherial layer. The order is divided into three families on the basis of conidia and conidiophore formation.

(1) *Pythiaceae.* In this family the sporangia are formed at the ends of ordinary hyphae. These produce swarm spores. Oogonia are large, thin-walled, globular bodies. The antheridia are small, elongated bodies borne on the same, or adjoining, mycelial threads. The important genera are *Pythium* and *Phytophthora.*

Pythium

Zoospores are produced in sporangia that are not differentiated but are united to the rest of the mycelium. Mycelium abundant. Sporangia germinate through a beak which permits the contents to escape as a thin-walled vesicle containing laterally biciliate zoospores. In some cases the sporangia may germinate by a direct rupture of the wall.

Oogonia are similar to the sporangia in size and structure. They contain many nuclei. As the oogonia develop toward maturity the nuclei migrate to the outer margin and degenerate, except one, which functions as the egg. The antheridia are smaller, more elongated and borne on smaller branches which are either on the same one, as the oogonium, or on a neighbouring one. On coming in contact there is a penetration of the oogonium wall by a tube from the antheridium. The male passes over into the oogonium and fertilization results after which the oogonium becomes a heavy-walled resting spore or zygote.

Phytophthora

Zoospores may or may not be present. If present they are produced in conidia borne on branched conidiophores. They are more or less lemon-shaped with a papillate point. When mature they may contain zoospores, in which case they are zoosporangia. In some cases they germinate directly. The mycelium is coenocytic, except when the gametes are formed. Conidiophores emerge from the stomata singly or in groups. There are little thickenings just below the point where the apparently lateral conidia are attached. This is an important character of identification.

(2) *Albuginaceae*. Mycelium fine, intercellular, feeding by means of globular haustoria. Conidiophores club-shaped and, together with the conidia, produced in dense sori, or blister-like pustules. From the character of the sori the name "white rust" has been given the fungus. Sexual reproduction by heterogametes. Antheridia and oogonia are formed on adjacent hyphae off the internal mycelium. A single genus.

Albugo

Conidia produced by successive budding off of the end of the conidiophore. Sori white. Hypertrophy of leaf, stem and fruit. Antheridium and oogonium on adjacent hyphae. Antheridium multinucleate. Only one unites with the egg. Balance disintegrate. Oogonium may be multinucleate in which case a male gamete unites with each egg nucleus. The zygospore is a heavy-walled spore which may bear ridges or knobs. The zygote must remain in the host tissue until the host plant decays. The zygospore germinates by cracking the wall permitting the contents to emerge as a sack in which are a number of zoospores. Upon the maturity of the sack these are liberated and may then infect another host plant.

(3) *Peronosporaceae*. Conidiophores are erect, simple or branched, generally emerging in groups from the stomata. Conidia produced one on a branch at a time, rather than sporangial production as in the case of *Pythiaceae*. Oospores globular in shape, containing from one to many nuclei but the mature ogonium contains a single egg. Antheridia are also multinucleate, but there is only one union between male and female nuclei. There are five important genera. *Sclerospora, Plasmopora, Pseudoperonospora, Bremia and Peronospora.*

Sclerospora

This genus is characterized by a large number of oospores and comparatively few conidia and conidiophores. Mycelium much branched with small vesicular haustoria, bearing the conidiophores erect either solitary or in groups of two or three. Oogonia are elliptic, or globose elliptic, hyaline, smooth, and are produced within the tissues of the host plant. Zygotes are brown with irregular wrinkles on the surface. The epispore and oogonial wall are so closely united that they appear to be one.

Plasmopara

Characterized by the tree-like conidiophores. The branching is at right angles and monopodial with the dichotomous branching of many others. Conidia globose to ovoid, hyaline or smoky. Gemination is indirect, i.e., by zoospores, or direct.

Pseudoperonospora

Similar to the one above except that the tips of the conidiophore branches are acute rather than obtuse. Conidia may be lemon-shaped or egg-shaped. They may be gray or smoky in appearance.

Bremia

Sporangiophores dichotomously-branched, the branches ending in little disks or swellings. Germination of the conidia either direct or by swarm spores.

Peronospora

Conidiophores dichotomously-branched, at acute angles. The ultimate branches are acute. Germination of the conidia direct by germ tube.

3. *Zygomycetes*

The *Zygomycetes* are similar to the *Oomycetes* in having coenocytic mycelium but differ in that they have isogametes instead of heterogametes. Only one order is of importance.

a. *Mucorales*

The thallus body is composed of aerial hyphae. Sporangia are borne on slender hyphae. Sex organs are alike as to appearance but differ in that they must be of opposite sex potentialities in order to form a union. The order is divided into two families on the basis of sexual reproduction.

(1) *Mucoraceae.* Produce their spores in typical sporangia containing a columella. Important genera *Mucor* and *Rhizopus.*

Rhizopus. Fungus spread by stolons and the sporangiophores arise at the nodes of the stolons.

Mucor. No stolons are formed.

(2) *Choanephoraceae.* Sporangia of two kinds; those containing many spores and those containing a single spore. May reproduce by conidia. Important genera *Choanephora* and *Cunninghamella.*

Choanephora. Sporangia of two kinds. Macrosporangia globose, columella small, spiny, spores few. Sporangiophores simple, branched, erect. Microsporangia clavate, one spored stimulating conidia and borne on the enlarged apices of umbellately-branched sporangiophores.

Cunninghamella. Conidiophores erect, branched, terminating in spherical heads, furnished with small swellings which are points of conidial attachment. Conidia spherical, oval, often with irregular outline. The external membrane spiny with needle crystals. Zygotes globose, warty.

D. *Ascomycetes*

The class of fungi known as the *Ascomycetes* is one of the largest of the *Thallophyta.* It includes upwards of 15,000 species, many of which are parasitic upon important crop plants. The mycelium of the *Ascomycetes* consists of a much-branched, septate hyphae, which may be found within the host tissue or only on the surface. Assimilation is by means of haustoria.

The characteristic feature of the class is the ascus, a sack-like body, in which spores are produced, commonly called ascospores. The ascus varies in shape and size according to the species of fungus. In some the ascus is nearly round (the yeasts), in others it is elongated and narrow (brown rot of stone fruits) in still others it is nearly rectangular (peach leaf curl) and in some (*Nectria*) it is ovate. The ascospores vary in shape as much as the asci. They are usually elliptical in outline, but may be spherical or globose, even long and narrow.

The ascocarp is composed of many strands of hyphae may be merely a more or less flat surface (peach-leaf curl), a disk-shaped structure (brown rot of stone fruits), a globular body containing an opening for spore escape (Nectria), or the body may be completely closed as in the case of the powdery mildews.

The *Ascomycetes* are sub-divided into two subclasses, based upon the formation of an ascocarp.

1. *Hemiascomycetes*

In this subclass the asci are formed on the mycelium directly and without the formation of an ascocarp. The *Hemiascomycetes* are further subdivided into two orders on the basis of the formation of the ascus.

a. *Endomycetales*

In this order the zygote changes directly into an ascus. In some cases there is little, if any mycelium. There are three families in the order all based on the ascus formation.

(1) *Ascoideaceae.* The asci contain more than 8 ascospores. Gametangia may be multinucleate.

(2) *Endomycetaceae.* The asci contain 8 ascospores. Gametangia are uninucleate. Asci on a well-developed mycelium.

(3) *Saccharomycetaceae.* No mycelium is found. Reproduction is by budding. Asci are the product of a single cell. There are two genera that are more or less common, *Saccharomyces* and *Zygosaccharomyces.*

Saccharomyces. This genus contains the yeasts of industrial importance. They are characterized by the asexual form of reproduction known as budding and by the formation of sexual spores without any evidence of a sexual process.

Saccharomyces cerevisiae is the commonly used yeast in brewing and baking.

Zygosaccaromyces. These reproduce by budding and spores are formed by conjugation. The number of ascospores varies with the different species, but is usually four. Conjugation cells usually of equal size.

b. *Taphrinales*

The *Taphrinales* differ from the *Ascomycetes* in the succeeding orders by having the asci produced without any definite hymenium. The order is divided into two families on the basis of the behaviour of the gametangia, *Protomycetaceae* and *Taphrinaceae.*

(1) *Protomycetaceae.* The resting cells chlamydospores) are thick-walled.

The spore mother cells along the wall of the gametangium form four spores each. When the chlamydospore germinates the exospore splits and the contents (endospore) emerges to form a large many-spored sack. The common genus is *Protomyces* which causes galls on various species of plants.

(2) *Taphrinaceae.* In this family the resting spores are thin-walled. When germination occurs the germ tube emerges from the host tissue, cuts off a terminal cell and forms an 8-spored ascus. This becomes many-spored by budding. The common genus is *Taphrina.*

Taphrina. The members of this genus possess an annual or perennial mycelium and a typically 8-spored ascus. The asci are borne on the surface of the infected parts of the plant. In the past the ascus shape has been used to set up some three sub genera. Although the ascus is typically 8-spored, due to budding, many more may appear. The common disease is peach leaf curl.

2. *Euascomycetes*

The members of the sub-class *Euascomycetes* differ from those of the *Hamiascomycetes* in that the zygote first develops the ascogenous (ascus forming) hyphae and then produce the asci. Martin (42) has classified them all under the *Euascomycetes.* Older text-books will refer to the sub-classes *Discomycetes* and *Pyrenomycetes.* But the tendency now is to list them all under the one sub-class.

a. *Eurotiales*

These forms bear the asci either singly or in tufts without an extensive stroma. The interior of the ascocarp is filled with the asci and ascogenous hyphae.

The order is divided into four families on the basis of the peridium (ascus wall) and ascocarp structure.

(1) *Gymnoascaceae*. Peridium is composed of loosely woven hyphae.

(2) *Eurotiaceae*. In this family the peridium is pseudoparenchymatous (false parenchyma made of strands of hyphae). The ascocarp is sessile.

Aspergillus. Hyphae and conidiophores are erect and more or less colourless. Conidiophores unbranched and bear at the tips a swollen knob on which the conidia are arranged in chains. The asci are rarely found.

Penicillium. These are the blue moulds. The hyphae are creeping but the conidiophores are erect. They are branched and the tips are again divided so that they resemble broom straw sorghum in branching. *Penicillium digitatus* and *P. italicum* have been shown to be responsible for much of the loss, sustained by the citrus growers, due to storage rots.

(3) *Onygenaceae*. In this family the ascocarp is stalked, capitate, of medium to small size and with an opening in the upper side.

(4) *Elaphomycetaceae*. In this family the ascocarp is sessile, matures below the surface of the soil and does not break open. It is medium to large in size.

b. *Myriangiales*

These forms differ from the above-mentioned orders in having a well-developed stroma which is often gelatinous in nature. The order is divided into families in accordance to the place of origin of the asci. Spores are liberated at the decay of the ascocarp.

(1) *Atichiaceae*. The asci arise at various levels.

The thallus is gelatinous, superficial on leaves, more or less yeast-like. Found growing on insect excretion.

(2) *Myrangiaceae.* This family differs from the above in not having a superficial thallus or yeast-like cells. The stroma is massive and homogenous without a cover.

(3) *Elsinoaceae.* This family has a stroma composed of a gelatinous interior with a hard outer wall or rind.
One genus *Elsinoe.*

Elsinoe. The ascocarp is more or less covered and without a shield. Conidiophores are cylindrical with sharp-pointed apices.

(4) *Saccardiaceae.* There is no covering for the storma in this case.

(5) *Dothioraceae.* The members of this family possess an outer casing of the stroma. Locules are embedded in the stroma and the tissue between the chambers is composed of pseudo (false) paraphyses.

c. *Dothidiales*

The members of this order possess a hymenial layer on which the asci are produced in tufts. The locules are more or less spherical, resembling perithecial cavities but without definite perithecial walls. There are five families in the order, the division being based on the stromatic formation.

(1) *Capnodiaceae.* In this family are found some of the sooty moulds. They have an extensive stroma which is much-branched. The ascocarps are borne singly at the tips of branches resembling perithecia. Most of these fungi are found associated with insect excretion. Only one genus is common.

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Capnodium. Mycellium black, cobwebby The hyphae often appear to agglutinate into skeins. Perithecia are superficial and composed of a cobwebby mass of hyphae. They are soft, fleshy or they may be slimy, cartilaginous to leathery. No ostiole present in the ascocarp. Asci typically 8-spored. Pycnidia may be present.

(2) *Pseudosphaeriaceae.* In this family the stroma is not as branched as in the above. It may aggregate into perithecia-like growths. These may be saprophytic or parasitic. The stroma usually forms a locule with an ostiole.

(3) *Corynelliaceae.* In this family the stroma is lobed. Each lobe contains a single locule which opens wide. The members of this family differ from those of the preceding in having several locules within the stroma.

(4) *Dothidiaceae.* The stroma is not lobed and the locules are embedded. The stroma is superficial.

(5) *Phyllachoraceae.* This family contains members with the stroma embedded in the host tissue. Otherwise similar to the above.

d. *Hemisphaeriales*

In this order the fruitification is a more or less disc form structure. The locules have no ostiole but rupturing irregularly. There are six families in the order.

(1) *Stigmataceae.* In this family the stroma is beneath the surface of the host tissue with a very scanty mycelium.

(2) *Polystomellaceae.* The family differs from the above in having a superficial stroma. The mycelium, which is largely internal, forms as a hypostroma.

(3) *Micropeltaceae.* (*Hemisphaeriaceae*) In this family the mycelium is internal but scanty.

(4) *Microthyriaceae.* In this family the stromatic layer is radically arranged. Very little if any, superficial mycelium.

(5) *Trichopeltaceae.* In this family the stromatic layer is parallel or radical. It is flat and thin.

(6) *Trichothyriaceae.* No stroma in this family. Mostly parasitic on other fungi.

e. *Erysiphales* (*Perisporiales*)

In this family there is no opening to the ascocarp. This type of ascocarp is called a cleistothecium. The asci are borne in the ascocarp in the form of an umbel and are free at maturity. There are three families under the *Erysiphales.*

(1) *Erysiphaceae.* Mycelium white. Cleistothethia bearing appendages.

Key to Genera of *Erysiphaceae.*

1. Asci one in each ascocarp.

Appendages indeterminate, flexuous, mycelium-like, unbranched.
. . . . . *Sphaerotheca*

Appendages determinate, dichotomously-branched at the apex
. . . . . *Podosphaera*

2. Asci more than one in each ascocarp.
Mycelium wholly external on the host.
Appendages various, mostly unbranched, hypha-like
. . . . . *Erysiphe*

Appendages hooked or coiled at the tip. Rigid, unbranched
. . . . . *Uncinula*

Appendages several times dichotomously-branched at the tip, rarely simple . . . . *Microsphaera*

Mycelium possessing haustoria that penetrate the host tissue by way of the stomata.

Appendages rigid with the bulbous base, unbranched. . . . . *Phyllactinia.*

(2) *Meliolaceae.* Mycelium dark. No gelatinous formation in the mycelium or upper part of the perithecium. One genus, *Meliola.*

Meliola. A dark sooty mycelium. The main difference between *Meliola* and *Erysiphaceae* is in the colour of the mycelium.

(3) *Englerulaceae.* The upper portion of the perithecium is gelatinous.

f. *Hypocreales.*

In this order the fruitification mass is soft and usually bright coloured. Such colour as red, white, scarlet, violet, yellow, blue, etc. Two families are set up on the character of position of the perithecia.

(1) *Nectriaceae.* In this case the perithecia are superficial.

(2) *Hypocreaceae.* In this family the perithecia are entirely imbedded in a stroma or stromatic base.

Nectria. The perithecium is soft and membraneous. It may be red, brown, single or grouped on the substratum and may be in or on a fleshy stroma. Ascospores are two-celled, ellipsoid and may be either pointed, or blunt. Conidia varied.

Martin (421) divides the *Hypocreaceae* into four tribes on the stroma character as follows:

Tribe *Nectrieae.* No stroma. *Nectria.*

Tribe *Creonectreae.* Stroma present *Creonectria.*

Tribe *Hypocreae.* Stroma seated directly on a substratum, usually patellate or effused, rarely clavate and erect. *Hypocrea, Hypomyces.*

Tribe *Cordycipiteae.* In this tribe the stroma arises from a sclerotium which is club-shaped and erect. *Cordyceps, Claviceps.*

g. *Sphaeriales*

The fungi of this order have a typically ostiolate, more or less globular perthecium with well-developed walls. The order has been divided by Martin (421) into two groups on the basis of the position of the perithecia, i.e., whether wholly or partially immersed. There are fifteen families.

(1) *Chaetomiaceae.* Perithecial walls carbonaceous. Hairy.

(2) *Sardariaceae.* Perithecia naked on only few hairs.

(3) *Sphaeriaceae.* Mouth of the perithecia papilla like.

(4) *Ceratostomataceae.* Perithecia with beak long and often hair-like.

(5) *Cucurbitariaceae.* Perithecia in tufts exposed at maturity.

(6) *Amphisphaeriaceae.* Base of the perithecium immersed. Ostiole circular.

(7) *Lophiostomataceae.* Ostiole is compressed, elongate.

(8) *Mycosphaerellaceae.* In this family the perithecia are immersed in a stroma or under a stromatic layer. The perithecial mouth mostly papillate (the mouth being soft and more or less superficial) *Mycosphaerella*, *Venturia*, *Physalospora.*

Mycosphaerella. Over 500 species. Perithecia subepidermal, barely appearing when mature, globular, lens-shaped, walls thin, ostiole flat or with very short neck. Asci club-shaped or cylindric, typically 8 spored. The spores are hyaline or greenish, more or less elliptical, 2-celled. No paraphyses are present. The imperfect form may be a *Ramularia*, *Aschochyta*, *Septoria*, *Pleospora*, *Cercospora*, *Ovularia*, *Cylindrosporium*, *Phyllosticta* *Phoma*, *Diplodia*, or *Septogloeum.*

Venturia. Perithecia are superficial or break through the epidermis at maturity. They are covered with bristles, possess an ostiole, are membraneous, dark-coloured. The asci either sessile or with short stripes. They are ovate or saccate. The spores are oblong to ovoid elliptic, hyaline or yellowish. The conidial stage is a *Fusicladium.*

Physalospora. Perithecia are subglobose and covered with a black membrane, may be coriaceous. The ostiole breaking through the membrane. Asci are club-shaped to cylindric. Spores ovoid or oblong, hyaline or subhyaline. Paraphyses present.

(9) *Gnomoniaceae.* The perithecia are usually beaked.

Glomerella. Stroma variable, usually definite, perithecia membraneous, generally pale ashy within. The beak is cylindric or filiform. The asci are fusoid. The spores are fusoid to sub-elliptic, 2-celled, hyaline. The conidial stage may be a *Phoma*, *Cytospora*, etc.

(10) *Clypeosphaeriaceae.* The stroma is a shield-like structure covering the perithecia. This structure is referred to as a clypeus. Hence the name.

(11) *Valsaceae.* The stroma is not a shield but is composed wholly of fungus elements. The conidia are produced in cavities in the stroma.

(12) *Melanconidiaceae.* Similar to the *Valsaceae* except that the conidia are borne superficially on the surface of the stroma.

(13) *Diatrypaceae.* The ascospores of the family are cylindrical, sausage-shaped and vary from hyaline to yellow brown. Little stroma is present in many cases. Perithecia may be superficial or under bark.

(14) *Melogrammataceae.* Ascospores 1—many-celled. Conidia typically in hollow chambers in the stroma. Hyaline to brown. *Endothia.*

Endothia. The perithecia are arranged in groups in circular order with the beaks pointed toward the centre. Spores are two-celled, hyaline.

(15) *Xylaria.* In this family the conidia are borne on a superficial layer on the surface of the stroma. Ascospores 1-2 celled dark.

h. *Laboulbeniales*

In the *Laboulbeniales* the stromatic wall is bright coloured soft and fleshy. These are mostly parasitic on insects or spiders. They possess a basal cell which functions as a haustorium. There are three families listed under the order—

(1) *Ceratomycetaceae.* No specialized antheridia. The spermatia are borne on the outside of

specialized branches of appendages.

(2) *Laboulbeniaceae.* Antheridia are present in this family. They are unicellular, flask-shaped structures.

(3) *Peyritschiellaceae.* Antheridia are compound in this family. That is several cells discharge into a common cavity from which they later escape.

i. *Hysteriales*

In this order the ostiole is an elongated slit. The perithecium is flattened, elongate, with the asci in a flat basal layer. There are four families in this order. These are based on the position of the ascocarp with reference that the host tissue.

(1) *Dichaenaceae.* In this family the ascospores are at first immersed in the host tissue and then erumpent. The ascocarp walls are tough leathery and black.

(2) *Ostropaceae.* The walls of the ascocarp are gray, or black, thick and corky.

(3) *Hysteriaceae.* The ascocarp walls are black, carbonaceous and either round or elongated.

(4) *Acrospermaceae.* The ascocarps are brown, club-shaped and with a tough membrane.

j. *Phacidiales*

In this order a membrane covers the hymenium layer until the maturity of the ascospores. The membrane then splits to permit the escape of the spores. During the splitting of the membrane the membrane becomes shaped as a star or some other irregular shape. The order is divided into three families on the basis of the ascocarp character.

(1) *Stictidaceae.* In this family the ascocarps are soft, fleshy and bright-coloured.

(2) *Tryblidiaceae.* In this family the ascocarps are carbonaceous black. They are at first immersed and then erumpent.

(3) *Phacidiaceae.* In this family the ascocarps remain imbedded in the host or stroma tissue.

k. *Helotiales*

The members of this order differ from those in the previous one in that the membrane does not split into a stellate form. There is no operculum. Asci escape through a pore.

(1) *Geoglossaceae.* In this family the ascocarps are club-shaped or pileate with a hymenium over the convex upper portion.

(2) *Patellariaceae.* In this family the ascocarps are discoid or saucer-shaped. The apothecia are leathery, horny, cartilaginous or gelatinous.

(3) *Mollisiaceae.* Ascocarps are discoid but the peridium is rounded or angular. It is thick walled and dark with the cells forming a pseudoparenchyma.
Pseudopeziza. Ascocarps fleshy, waxy rarely membranceous, at first sunken in the substrate, later erumpent. Ascocarps are disc-shaped and stalked. Asci 8-spored, elliptical or fusiform. Unicellular.

(4) *Helotiaceae.* Ascocarps are discoid same as in the above family but with an elongate, bright-coloured peridium composed of thin walled hyphae in parallel strands.
Sclerotinia. Sclerotia black and borne upon or within, the host tissue. Apothecia are disc-shaped and stalked. Asci are 8-spored, elliptic or fusiform and unicellular.

l. *Pezizales*

In this order the asci are operculate. The ascocarps are epigeic (develop above the ground) at least when mature. Hymenium is usually exposed before the maturity of the spores. There are four families in the order.

(1) *Cyttariaceae.* Mostly parasitic. In this family the ascocarps are pear-shaped with many pits.

(2) *Pezizaceae.* Saprophytic. The apothacia are cup-shaped or discoid and may be sessile or stalked.

(3) *Helvellaceae.* Ascocarps pileate and stipitate or columnar.

m. *Tuberales*

The asci in this order possess operculate covers. The ascocarps develop below the surface of the earth.

(1) *Tuberaceae.*

E. *Basidiomycetes*

The Basidiomycetes are among the most numerous of the fungi. At this time there are over 100 species of smuts and over 450 species of rusts recorded on plants in India. More smuts and rusts have been reported in India than *Ascomycetes.* Many of the smuts and rusts are parasitic upon the important crop plants.

The most important difference between the *Basidiomycetes* and the *Ascomycetes* is in the formation of the spores. In the *Ascomycetes* they are formed in a sac (the ascus) whereas in the case of the *Basidiomycetes* they are produced on aerial hypae, the basidia much as the conidiophores and conidia of the *Phycomycetes* and *Ascomycetes.* Conidia are not prominent among the *Basidiomycetes.*

The mycelium is septate and found mostly within the host tissue. Smuts are more or less systemic whereas rusts are much more localized. The mycelium may be classed under three heads—

Primary—1

That which comes from a binucleate sporidium, direct.

Secondary—2

That which comes from a binucleate sporidium, only one of the nuclei going into the mycelium.

Tertiary—3

That which arises from a binucleate sporidium as a result of a rapid mitosis but with no cell walls laid down between nuclei for some time later.

Sex organs are not well developed in the *Basidiomycetes*. Some Mycologists (Clement and Shear (123) for example) would derive them from the Ascomycetes. Others have considered that they are more closely related to the red algae. In the smuts copulation may be between adjacent cells of a hyphal thread, or copulation may take place between two neighbouring cells of different hypae. In either case the gametes are similar and are similar to the isogametes of the Phycomycetes.

The most characteristic reproductive structure of the *Basidiamycetes* is the basidium. This structure is produced by the germination of the chlamydospore of the smuts or the teleutospore of the rusts. At the time of the formation of the basidium the resting spore is binucleate. But, either just before or at the time of germination, the two nuclei fuse and then ameiotic division takes place followed by a mitotic division which gives rise, theoretically, to an equal number of basidiospores of each sex group. The two nuclei within the resting spore have not actually been called male and female but they carry the potentialities which will give rise to the male and female organs. In the rusts it is typically two. The same is true of the smuts but the number in each sex group is much more variable than in the rusts. Hanna (224) suggested that there might be as many as four sex groups in *Ustilago zeae*. Each of the sporidia representing one of the sex groups must be mated with a sporidium of another sex group. It cannot mate with one like itself. Thus if there are only two sex groups represented among the basidiospores of a rust and two of the same sex group fall upon the leaf of a host plant, infection will take place but no spores will form. Cragie (133) was the first to report upon the function of this phenomenon among the rusts and Hanna first reported it among the smuts. An excellent article on the question of the genetics of the smuts is that by Christiansen and Rodenhiser (126). It is suggested that every student should read this article. In an earlier issue of the same periodical Read (650) has also given an excellent review covering the physiologic specialization of fungi. More recently Read (651) has given us an article on the Physiologic Specialization of the Parasitic Fungi II which brings us uptodate. The student is also advised to read the articles by Walker (915), Fulling (232) Ausemus (38) and Wingard (933) which bear on the subject of the nature of disease resistance and parasitology. In India a number of workers have been contributing to the knowledge of the rusts and smuts, Dr. B. B. Mundkur and Dr. K. C. Mehta being among the most active.

Martin (421) divides the Basidiomycetes into two groups on the basis of the basidium character and the manner of germination of the basidiospores.

1. *Heterobasidimycetes*

In this subclass the basidia are septate or deeply divided. The basidia arise from chlamydospores. The basidiospores often germinate by repetition, that is by producing a stalk upon which another spore, similar to the first one, is produced. The secondary spore is forcibly discharged and may in turn germinate as the first one. The basidiospores may also produce conidia. The class is divided into three orders on the basis of the basidiocarp (a body specialized for the production of basidia).

a. *Tremellales*

The basidiocarp usually well-developed. It may be gelatinous, waxy or coriaceous. For the most part they are saprophytes but may be found on mosses and vascular plants. There .are eight families in the order separated on the basis of the basidia formation.

(1) *Tulasnellaceae.* Epibasidia (an outgrowth of the probasidum upon which one or more basidiospores are borne) enlarged, spore-like and cut off by a septum from the hypobasidium.

(2) *Dacrymycetaceae.* Epibasidium not as in the preceding family. The probasidia (the young stage of a basidium up to the time the sterigmata begin to develop) are cylindrical to narrowly clavate. The basidia are not septate but become forked by the formation of two epibasidia on each of the hypobasidia tips.

(3) *Sirobasidiaceae.* The basidia in this family are septate and not forked. The probasidia are subglobose, ovate or pyriform, are rarely broadly fusiform. The basidia occur in chains. No epibasidia or sterigmata.

(4) *Themellaceae.* The members of this family are similar to the above but the basidia are not found in chains (catenulate).

(5) *Hyaloriaceae.* These have a semi-membrane covering the basidiocarp. The spores are retained within a gelatinous sheath.

(6) *Phleogenaceae.* These forms have a complete covering over the basidiocarp. Basidiospores are sessile.

(7) *Septobasidiaceae.* In this case the basidiocarp is naked. The basidiospores borne on a sterigmata. Often found parasitic on scale

insects. May be found on woody plants as well.

(8) *Auriculariales.* These are saprophytes, mostly on vascular plants. Sometimes on mosses or fungi.

b. *Uredinales*

In this order the basidiocarp is formed by a mass of probasida, which may or may not have a peridium. There may be additional spore forms present. All are parasitic on vascular plants. The members of this order are usually known as "rusts". They are among the oldest known plant disease fungi. Centuries ago men wrote about the destruction of plants wrought by blights and blasts, the wrath of the angry gods, evil spirits and other unexplained agencies. It is now believed that much of this destruction was caused by the rust fungi but as the microscope was then unknown, it was not possible for them to determine the true cause. It was not until 1865 that the first life cycle of a rust was worked out. At that time Anton de Bary published his observations on the life cycle of the black stem rust of wheat and opened the way for the study of other rusts which were observed but not understood.

The *Uredinales* are distinguished from the smuts by the character of the basidium. The number of spore forms differs with the different species and this is another differentiating character. Symbols have been set up to represent the various spore forms and it is possible, by means of these symbols, to write the spore formula of the species. The symbol O indicates the presence of male and female gametes. The numeral I refers to the aecidiospore stage. The numeral II refers to the uredospore stage and the numeral III refers to the teleutospore stage. The numeral IV refers to the basidia. Thus a rust with the formula 0, I, II, III, IV will possess all of the spore forms. One with the formula I, II, III, will possess the aecia, uredina and telia stages. Many of the rusts have only the II, III, IV stages known.

The pycnia, (spermagonia) are minute flask-shaped structures which open to the air so that the spores may escape. They are just beneath the epidermis, mostly on the upper surface of the barberry leaves, with the neck protruding through exposing the ostiole. The pycnia are of two sorts of hyphae, the outer being sterile and the inner fertile.

Two families will be discussed.

(1) *Melampsoraceae.* The teliospores are sessile, forming crusts or cushions or cylindrical masses (as in linseed rust). The sori (fruiting masses) are in the mesophyll or epidermis of the host. The family is divided into four genera. *Cronartium*, *Melampsora*, *Coleosporium* and *Cronartium*. They will be briefly discussed.

Cronartium. A heteroecious group whose life cycle of development includes pycnia and aecia on the trunk and branches of members of the coniferous genus *Pinus.* The uredia and telia occur on the various herbacious and woody dicotyledons. *Cronartium ribicola*, the white pine blister rust of the temperate regions, is an example.

Melampsora. This genus contains both autecious and heteroecious species of rusts. In the case of the heteroecious species the telia are on woody plants. An example would be *Melampsora medusae* with the telia on members of the genus *Pinus.* The common example of an autecious species is *Melampsora lini* with the entire life cycle on the linseed plant. The pycnia are subepidermal or subcuticular without paraphyses.

Coleosporium. Heteroecious rusts whose life cycle of development includes pycnia and aecia on the leaves of the coniferous genus *Pinus* and the uredia and telia on herbaceous plants.

(2) *Pucciniaceae.* The teliospores are stalked and in some cases are held together in a gelatinous matrix. The family is the most important one from the standpoint of the number of species parasitic on the important farm crop plants. Arthur (31) lists 17 genera, even when combining *Uromyces* and *Puccinia* into one, that contain important parasitic forms. The genus *Puccinia* is the most important one, containing nearly one half of all the known species of rusts. Arthur lists 650 species of *Puccinia* alone. Butler and Bisby (96) list 145 species as having been reported in India and Mundkur (517) lists 25 more, making a total of 170 known in India. Four genera are common in India. These are, in order of numerical importance, *Puccinia*, *Uromyces*, *Phragmidium* and *Gymnosporangium*.

Puccina. The teliospores are pedicelled (except for one species), 1-2 celled, rarely more, smooth or vericose, oblong to clavate, usually with a thickened apex, firm pedicel and with the pores in the apex. Aecia are similar to the sori with catenulate spores covered with a periderm.

Uromyces. In this genus the teliospores are one-celled. Arthur (31), however, contends that this is not a constant character and therefore does not constitute a good character. He unites the two genera, *Uromyces* and *Puccinia* under *Puccinia.* However, the two are separate in much of the literature and for that reason are treated as two genera in this book.

Phragmidium. This is an autecious genus. Butler and Bisby (96) list 15 species in India. There is no peridium (the outer coat which covers the spores) and the aeciospores are catenulate. Teliospores are large and conspicuous.

c. *Ustilaginales*

Basidiocarp much the same as for the preceding order but the epibasidia may be septate or not with the basidiospores sessile. The spores germinate like yeast cells (by budding) or, as in some cases, by repetition. The teleutospores rarely germinate to form a mycelium directly. The spore masses are usually black and it is this character that has given the name "smuts" to the members of the order. The *Ustilaginales* are divided into three families on the basis of the type of fructification.

(1) *Graphiolaceae*. In this family the fruiting body is a cup-like structure with a relatively thick peridium. The chlamydospores (teliospores) are in chains which are separated by sterile hypha. These are found on palms in the warm climates.

(2) *Ustilaginaceae*. The fruiting structure is not cup-shaped. It is composed of a mass of chlamydospores, some of which may be sterile. The pedicel sheath may be sterile and either present or absent. The chlamydospores, on germination, give rise to a transverse, septate epibasidium which produces a number of basidiospores, usually 4. The chlamydospores rarely form hyphae directly. There are four important genera in the family. *Ustilago*, *Sphacelotheca*, *Sorosporium* and *Tolyposporium*.

Ustilago. These fungi possess no peridium about the sorus. The spores are exposed as a black mass which is rusty at maturity. They are single, globose, finely verrucose or spiny and germinate to produce a promycelium directly. The common loose smuts of corn, oats, wheat and barley belong to this genus.

Sphacelotheca. These smuts infect the blossoms and especially the ovaries of the plant, the kernel being replaced by a mass of smut fungus which is surrounded by a false membrane. Within the mass is a central column, or columella, which is sterile. The spores are single and mostly of a reddish brown colour. The grain smut of jowar is common example. *Sorosporium.* Like *Sphacelotheca,* but without the false membrane about the sorus. At first the spores are held together by a gelatinous material but soon after maturity this disintegrates and they remain only loosely held together.

(3) *Tilletiaceae.* The fructification is similar to that above but the chlamydospores germinate to form a non-septate epibasidium that bears a cluster of slender basidiospores at the tip. Two genera. *Tilletia* and *Urocystis.*

Tilletia. The fungus replaces the inner portion of the kernel and a smut ball is harvested instead of a normal grain. When the grain is crushed the spores are dusty and scattered over the rest of the grain by which means they are distributed. These are the bunts of wheat and rice. They are characterized by an odour, similar to fish, when the grain is badly infected.

Urocystis. The spores of this genus are held together in balls, each ball having a layer of sterile, more or less hyaline, cells around the outside.

Entyloma. In the older textbooks another genus is often listed under the *Tilletiaceae.* The members of the genus *Entyloma* have the sori imbedded in the host tissue. The spores are single rather than in balls.

2. *Homobasidiomycetes*

In this subclass the basidia are simple, cylindrical, uniform or broadly clavate. There is no specially differentiated probasidium. The subclass is divided into 7 orders on the basis of basidiocarp and hymenium characters. These are the true basidiomycetes and include the mushrooms and toadstools. Although they cause a great deal of damage to woody plants, few of them are important on the crop plants of the farm. The characters of the orders, only, will be given here.

a. *Exobasidiales*

In this order there is no special basidiocarp. The diseased tissue of the host is found covered with a hymenium. They are mostly vascular parasites. *Corticium* (*Rhizoctonia*) is placed under the family *Telephoreceae* but, as it is discussed under the *Mycelia sterlia,* it will be omitted here.

b. *Agaricales*

Commonly called the "gill fungi" these are the mushrooms found on compost heaps and about manure piles. The basidiocarp is present and is in the shape of an umbrella. The hymenium is spread over the surface of gill-like structures on the surface of the pileus. Many of the agarics are edible. Some are extremely poisonous and unless the person is familiar with the various species it is better to leave them alone. This is specially true of the white-spored forms, as *Amanita muscaria* and *Amanita phalloides,* two white-spored species which are very poisonous, containing alkaloids which can cause death.

c. *Hymenogastrales*

In this order the hymenium is mostly lacking or very indistinct. The spore-bearing tissue (gleba) is waxy, sometimes becoming slimy and fetid when not exposed to drying. The order is divided into four families based on the development of the fruiting structure. These are puff-ball like.

d. *Phallales*

The gleba is slimy and fetid at maturity. These forms attract flies and in this way are scattered about. They are the common "stink horn" fungi and the fungus body is long and at first inclosed in a membrane.

e. *Lycoperdales*

In this order the gleba is dry and powdery at maturity. These are the true puff balls. The spores are commonly small and pale under the lens.

f. *Sclerodermatales*

In this order the spores form a powdery mass. The chambers remain together and also attached to the peridium. Spores are dark.

g. *Nidulariaceae*

In this order the gleba is waxy. The basidia are united into a hymenium which lines the walls of the cavities. These cavities remain separated at maturity from the cup-like peridium. The whole structure resembles a bird's nest and they are referred to as the "bird's nest fungi".

F. *The Fungi Imperfecti*

In this class the perfect or sexual stage is not known, or if known, it has been only recently discovered and the fungus has been listed among the *Fungi Imperfecti* for so long that it has been widely published in literature in that group and it is retained among them for simplicity. In the older textbooks three orders have been listed, namely, *Hiphales* (Moniales), *Melanconiales* and *Sphaeropsidales*. Some authors have listed the *Sphaeropsidales* as the *Phomales.* Recently Martin (421) has suggested the name *Phyllostictales* for the *Sphaeropsidales* and has added, as the fourth order, *Mycelia Sterila.* The classification according to him being, *Phyllostictales, Melanconiales, Moniliales* and *Mycelia Sterila.*

a. *Phyllostictales*

In this order the vegetative reproduction cells are borne on specialized hyphae (conidiophores) which are inclosed in a flask-shaped fruiting body (pycnidium) and which opens by a pore (Ostiole). The members of the order have been variously classified as *Sphaeropsidales* and as *Phomales.* The order has been divided into four families on the basis of the pycnidia formation. One family is of importance to the plant pathologist.

(1) *Phyllostictaceae* (*Sphaeropsidaceae*) The members of this family have pycnidia that are black, carbonaceous, membranous coriaceous and of varying shapes. The family is divided into a number of genera, the most important ones being, *Phyllosticta, Phoma, Diplodia, Septoria, Phomopsis, Aschochyta, Hendersonia* and *Stagnospora.*

Phyllosticta. These possess a stroma. The pycnidia are thin-walled, parenchymatous. Not sclerotium like. Spores single-celled, hyaline. Found mostly on leaves.

Phoma. Phoma possesses no stroma. Pycnidia thin-walled, erumpent. Mostly on stems. Spores single-celled, hyaline.

Diplodia. Pycnidia at first covered and then erumpent. Sub-globose, mostly papillate, thick-walled, formed of blackish brown parenchyma outside, Hyaline. Spores septate, brown.

Septoria. Pycnidia standing singly without stroma. Thin-walled or with many thin-walled cells. No beak on the pycnidium. Spores elongated, hyaline, filiform or linear, straight or flexuous. May be curved or worm-like. Often with guttules or septa.

Phomospis. Pycnidia lens-shaped, conical or more or less globose. Walls several cells thick. Below olivaceous in colour becoming darker above ostiole, spores hyaline, mostly fusoid with sub-acute extremities, but sometimes ellipsoid with narrowed ends or even oblong. Biguttulate.

Ascochyta. *Pycnidia* standing singly without any stroma. The spores are hyaline without appendages. Two-celled.

Stagnospora. Pycnidia single without stroma. Spores usually straight, oblong linear or elliptical, hyaline, guttulate. Two or more septa.
Hendersonia. Like *Stagnospora* except spores dark.

(2) *Nectrioidaceae.* The members of this family are the light or bright-coloured fleshy pycnidial forms.

(3) *Leptostrometaceae.* Members of this family have irregular or shield-shaped pycnidia which are black.

(4) *Excipulariaceae.* The members of this family have cup-shaped (closed at first, later open) pycnidia which are black.

b. *Melanconiales*

Conidia borne in acervuli, definitely circumscribed and finally free on the substratum. There is only one family *Melanconiaceae* with some 45 genera and 1,200 species. The most representative genera are *Colletotrichum*, *Gloeosporium*, *Pestalotia* and *Coryneum*. The order is divided into two groups on the basis of the spore colour. The genera containing the colourless spored forms being placed in the *Hyalospermae* and those with the coloured spores being in the *Phaeospermae*.

(1) *Melanconicaeae*. Mycelium internal. Conidiophores form a stratum typically bearing conidia in acervuli which are immersed or erumpent, black or light-coloured, waxy, corneous or even submembraneous, with or without setae. Conidia variable.

Colletotrichum. Setae (bristle like growths in the acervuli) distinguish this genus from the following one.

Gleosporium. There are no setae found in the acervulus. However, this is not always a reliable character to observe as they may not always be present in the acervuli of members of the genus *Colletotrichum*.

Pestalotia. Acervuli form beneath the host tissue surface and then rupture through it. They are convex or disc-shaped and are dark to black in colour.

Coryneum. The acervuli are disc-shaped or cushion-shaped. They are beneath the surface but erupting out to the surface. Black and compact. Conidia are oblong to fusoid. Septations 3 or more.

c. *Moniliales*

In this order the specialized hyphae on which the vegetative reproductive cells are borne, are not organized into a hymenium. For this reason they are considered to be among the most primitive of the *Fungi Imperfecti*. The order is divided into some six families on the basis of the hyphae development. Within the families the genera are based on spore sections according to the number of cells and the colour of the conidia.

(1) *Pseudosaccharomyces.* Very little hyphae but the cells do not reproduce by budding or by repetition.

(2) *Sporobolomycetaceae.* In this family the cells do reproduce by budding and by repetition similar to basidiospores. Martin (421) suggests that these are related to the *Tremelleles* and are the imperfect forms of these fungi.

(3) *Moniliaceae.* The fungi possess, generally clear or hyaline conidia and mycelium with no differentiation. The conidia may or may not be borne on specialized conidiophores. There are several important genera.

Verticillium. Hyphae creeping with erect conidiophores which are verticillately (whorled) branched. Conidia are borne singly at the apex of the branchlets, globose-ovoid, hyaline or light-coloured.

Botrytis. The hyphae are creeping with simple, more or less markedly dendritic branched conidiophores. The conidiophores are erect with various branchings. They are thin and apically pointed or they may be thick and obtuse. The conidia are variously grouped at the apex of the branches but never in true heads. The conidia are globose, elliptic or oblong, hyaline or light-coloured.

Piricularia. Conidiophores simple, rarely branched, conidia obclavate to pyriform, 2 to many septate, solitary, growing at the apex, hyaline.

Aspergillus. The ascocarps are small spherical smooth bodies which at maturity are filled with 8 spored asci. But the fungi have been included among the imperfect forms for a long time and as the perfect stage of many are not known they are retained in both the *Ascomycetes* and the *Fungi. Imperfecti.* The conidiophores are swoollen at the ends and bear numerous strigmata on which the spores are borne in chains. Mostly saproprytic but some are parasitic on plants and others cause bronchial trouble in man.

Penicillium. The same thing may be said of *Penicillium,* as was just said about *Aspergillus,* as regards its position in the classification. The characteristic conidiophores make distinguishing easy. In the case of *Penicillium* the tip of the conidiophore is not swollen but is branched repeatedly, more or less dichotomously branched. These branches bear the conidia in chains at the tips and in this they resemble a small broom.

Cephalothecium. The hyphae are prostrate with the conidiophores erect, simple, septate. The conidia are apical, subcepitate, oblong to pyriform, hyaline.

Ramularia. Conidiophores are in bundles, simple or with short, scattered branches, which may be flexuose, nodulose, or denticulate toward the apex, hyaline or light-coloured. The conidia are acrogenous (growing at the apex), hyaline, oblong, cylindric, typically many septate.

(4) *Dematiaceae.* Similar to the above family but the hyphae, or conidia, or both are dull coloured, brownish or black.

Heliminthosporium. The conidiophores are erect, rigid, subsimple, fuscous, conidia fusoid to elongate clavate or cylindric, pleuriseptate, dusky brown and smooth.

Alternaria. Conidiophores erect, subsimple, short conidia clavate to muriform, septate. Often borne in chains.

Macrosporium. Conidiophores fasciculate, erect or not, more or less branched and dark or coloured. The conidia are usually apically elongate or globose. They are dark-coloured.
Cercospora. Conidiophores are variable. In some cases they are very short while in others they are as long or longer than the conidia. They may be simple or branched. They are dark olivaceous in colour. The conidia are vermiform or filiform, straight or curved, multiseptate, subhyaline to dark.

Cladosporium. The hyphae are decumbent, much branched, olivaceous in colour. The conidia are globose to ovoid and greenish in colour.

(5) *Stilbaceae.* The conidiophores are aggregated together to form columns. or pillars, called coremia.

(6) *Tuberculariaceae.* The distinguishing character of the family is the sporodochium. The sporodochium is a structure composed of the aggregation of hyphae and conidiophores.

Tubercularia. The conidiophores are interwoven to form a fruiting layer over the surface of the sporodochium. It is straight or curved or may be broken up into single cells. Branches are elongate or short. Conidia terminal, seldom appearing laterally. Borne singly, usually ovate or elongate, cylindric or globose, seldom curved or boat-shaped. They are hyaline and cover the whole layer with a thick sheath. Sprodochia wart-like or cushion-like. Sessile or stalked, erumpent and often surrounded by the covering of the substrate. Sometimes with simple hairs about the margin.

Fusarium. The conidial layer cushion-shaped or somewhat extended without a definite limit. Conidiophores branched. The conidia are terminal at first, simple, spindle-shaped or sickle-shaped, many-celled with indistinct cross-walls. It is a large and complex genus. There are 65 species and 78 varieties with some 16 sections set up in "*Die Fusarium*" of Wollen-weber and Reinking (1935) (938).

G. *Mycelia Sterilia*

In this subclass, or subsection, the mycelium is sterile, i.e., does not produce conidia. It is not divided into families as the others as there is no very definite base on which to divide it. The group receives its name from the fact that the mycelia are unfruitful.

They may or may not produce sclerotia but in any case they depend upon the vegetative mycelium to carry them over from one season of activity to the next. Two genera are recognized.

Rhizoctonia. These fungi produce sclerotia that are connected by strands with the rest of the mycelium. It is often referred to as *Corticium* (which is the imperfect stage of *Rhizoctonia*) and that is probably the more correct term to use when it is listed under the *Mycelia Sterilia.* It is also sometimes referred to as *Macrophomina.*

Sclerotium. The sclerotia are rounded or irregular in form, cartilaginous-fleshy but are not connected to the rest of the mycelium. The cortex in this, is membranous. There are a large number of species, many of which are parasitic on economic host plants.

The Virus Diseases

One of the most serious problems which confront the plant pathologist to-day is that of the mosaic diseases caused by plant viruses. Although they are among the most destructive of the plant diseases known, their history dates only from the latter part of the 19th century. The first virus disease was reported in Germany as a "Mosaikkrankheit" in 1885. Sourauer (722) records the report of Van Sweiten who called attention in 1857 to the mosaic-like character of the chlorophyll pattern found in the leaves of tobacco in a Dutch plantation. In 1894 Iwanouski demonstrated that the causal organism of the disease was able to pass through a porcelain filter without reducing its viability. At that time there was a great deal of argument regarding the cause. Most of the plant pathologists of that time considered the causal organism to be bacterial in nature if not actually a bacterium.

But the discovery of the ability of the causal organism to pass through filters directed the study into a new phase. As each new worker took up the study, report after report came into print and the mass of literature increased by leaps and bounds. The reference list which has been selected for this section of the work has been selected mostly because it is representative of the type of reference which may be found in text and periodical.

Although no one to date has succeeded in isolating the causal agents of the mosaic diseases, this fact has not held up the work on such diseases. To-day workers in every land are busy studying means for the identification, classification and control of the virus diseases. Nor is this industry on the part of the plant pathologists merely one of academic interest for there is scarcely a single cultivated crop plant that is not attacked by at least one virus disease. Scarcely an issue of the leading plant pathology periodicals is received which does not contain an account of either a new virus discovered or some new fact about an old one being added to the knowledge we already possess.

DEFINITION

There have been many attempts at defining the virus diseases. The word "virus" means slime or poison. Funk and Wagnalls' Comprehensive Standard Dictionary describes a virus as "a morbid poison that is the medium for communicating infectious disease." Holmes (288) gives the following definition which seems more adequate:

"Etiological agents of disease, typically of small size and capable of passing filters that retain bacteria, increasing only in the presence of living cells, giving rise to new strains by mutation, not arising *de novo.*"

It should be mentioned that some viruses causing diseases cannot be filtered but they are included in the definition until they have become better known.

There is some possibility of confusion between the terms mosaic and virus. "Mosaic disease" is a direct translation from the German "Mosaikkrankheit" which was used to describe the disease of tobacco reported in 1885. But it soon became evident that this was only a symptom and could not be used to describe all forms of the new disease. Many of the manifestations of the disease were not mosaic-like in character at all. It was then that the term virus came into use. The word "mosaic" is used to-day to describe a symptom of the virus disease rather than a group of diseases and the group of diseases are referred to as virus diseases. Some have attempted to introduce the term "viroses" into use but this has not met with general favour.

Distribution

The viruses of plant disease are world-wide, having been reported in every country. The range of host plants is evidenced by the list given by Smith (717) which consists of 155 viruses on 50 genera distributed throughout the plant kingdom. Holmes (288) in his Handbook of Phytopathogenic Viruses, lists 129 distinct viruses. The difference between the lists offered by Smith and Holmes may be due to the fact that as Holmes' list came out four years later some of the viruses, which were considered as distinct at the time of the publication of Smith's text, had been combined with some others by the time Holmes' book was published. New viruses had been named but not as fast as recombinations had been made. Smith (717) in his Textbook of Plant Virus Diseases lists the following genera and the number of viruses known on each in 1935.

Genus.	No. Viruses.	
1. Delphinum	2	
2. Peoria	1	
3. Anemone	1	
4. Brassica	4	
5. Matthiola	1	
6. Beta	5	6
7. Pelargonium	1	
8. Passiflora	1	
9. Cucumis	3	
10. Gossypium	1	
11. Manihot	2	
12. Ribes	1	
13. Fragaria	4	5
14. Rubus	6	
15. Holodiscus	1	
16. Prunus	7	4
17. Pyrus	2	
18. Rosa	4	

Genus.		No. Viruses
19.	Phaseolus	3
20.	Soja	1
21.	Medicago	4
22.	Pisum	5
23.	Trifolium	1
24.	Robinia	1
25.	Arachis	1
26.	Ficus	1
27.	Humulus	4
28.	Santalum	3
29.	Vitis	1
30.	Apium	3
31.	Vaccium	1
32.	Dahlia	3
33.	Callistephus	2
34.	Lactuca	1
35.	Nicotiana	20
36.	Lycopersicum	6
37.	Hyocyamus	1
38.	Datura	1
39.	Solanum	18
40.	Ananas	1
41.	Musa	3
42.	Tulipa	1
43.	Lilium	1
44.	Allium	1
45.	Iris	1
46.	Fresia	1
47.	Saccharum	11
48.	Zea	3
49.	Triticum	2
50.	Oryza	2
	Total	155

This list is not complete for we know of other genera that are attacked by viruses. For example, there are species of the genera Hibiscus, Zinnia, Capsicum, Citrus, Carica, Eleusine and others which have been found with diseases caused by viruses. A number of these occur in India. Dastur (156) reported the mosaic of sugarcane in India in 1921. McRae (443) reported a virus on *Eleusine coracana* in 1929 and at the same time reported a rugose mosaic on sugarcane. Galloway (238) reported a virus disease on *Elletaria cardamomum* in India in 1935 which is not included in the list given by Smith above. Kulkarni (352) reported a virus on cardamon which killed the plants in three to four years.

Citrus species were found to have a mosaic-like disease in the Madras District in 1934. McRae (449) and Pal (468) reported the separation of the tobacco mosaic into types A, B, C, D, and X on the basis of morphological characters. Rao (647) reported a serious disease on *Santalum album* Linn, which caused a spiked condition of the plant. Sundararaman (764) reported a virus on *Dolichos lablab* in 1932 and Uppel (844) reported virus diseases on chillies and sunflowers in the Bombay Presidency. These are only a few of the reports to be found in the Indian literature on the presence of virus diseases.

Field crops on the Institute farm at Allahabad are attacked by a number of virus diseases. The symptoms are often such as to indicate that more than one virus is responsible for the disease. At the present time tomatoes, potatoes and chillies are all suffering from virus diseases which show complex symptoms and appear to be caused by at least two viruses. Maize shows symptoms which indicate that two or more viruses are present. On the beans, chlorotic leaves indicate that perhaps only a single virus is present and on papayas the leaf curl is all of one type and thus would indicate a single virus.

By symptoms here is meant the appearance of the disease on the host plant. This is a very indirect way of describing an organism but in this case we have no other recourse for we cannot see it and the thermal death point, the longevity in vitro, the effect of dilution and the filterability of the virus are of little value to the plant pathologists who want to be able to identify in the field the diseases caused on the crop plants. The symptoms of the virus diseases have been more or less classified and have been made use of in arranging the diseases in related groups. Holmes (288) has arranged the viruses in groups according to the symptoms produced on the host plants. His groups are as follows:

Yellow group. In this group are those viruses causing yellowing of the leaves and witches brooms through stimulation of normally dormant buds.

Mosaic group. In this group are included all those viruses which cause mottling or spotting of the leaves. Also those which cause necrotic spotting.

Ringspot group. In this group are those viruses which cause necrotic or chlorotic spotting which are associated with concentric rings, e.g., tobacco ring spot.

Fiji disease group. In this group are those viruses causing a proliferation of normally inactive tissues. No chlorotic or necrotic mottling. Fiji disease of cane is an example.

Spindle tuber group. In this group are those viruses causing increase in the number of sprouts or branches. These are long, slender and weak. Potato spindle tuber is an example.

Leaf curl group. The viruses in this group cause a dwarfing and curling of the leaves. The curling may take a number of forms, as crinkling, rolling, or crinkling as well as curling.

The difficulty with this type of grouping is the correct identification of the particular character. It is difficult to use for the characters are often masked by or confused with each other so that classification is uncertain. It does offer advantages, however, for it does not require more than good eye-sight and a familiarity with the group characters.

The Effect upon the Host Plant

There are many effects upon the host plant which are not visible to the naked eye or, if visible, may not be used as identifying characters. In general, the plants are dwarfed and this condition may be accompanied by a dilution of the chlorophyll on the one hand or an intensification of the green on the other. Sometimes the dwarfing may be systemic or again it may be only local and confined to a few branches. Dwarfing may be accompanied, as in the case of potato spindle tuber, with an increase in the number of stalks or branches. In other cases there may be malformation of the branches. In the case of peach mosaic, Hutchins et al (299) reported witches broom effects. Mosaiced tomatoes on the Institute farm at Allahabad often show thickened and deformed stems. In some cases they are flattened and twisted. In some cases the stems may bear streaks of light and dark colour. Johnson (318) reports a streak on tobacco which had been known for a long time but was identified as a virus only in 1936.

On the leaves there are numerous symptoms. It was the pattern of the chlorophyll of the leaf that gave the disease the name mosaic, as stated in an earlier paragraph. These areas are variable in colour and shape.

There is often a curling and a roughening of the leaves. In some cases there is a puckering of the leaves, produced by alternate raised and depressed areas.

There is often a curling and dwarfing together with distortion of the leaves as in the case of the tomato virus disease. Sometimes the leaf margins are dissected so excessively that they have been named "fern leaf". Chamberlain (102) recorded a virus on swedes in New Zealand that caused a blistering of the leaves. Larson and Walker (362) record a virus on cabbages which caused a clearing of the veins.

On the flower, the virus diseases cause several different effects. The flowers may be dwarfed, or malformed, or show a mottling of the corolla and falling of the blossoms as in the case of the tomato and potato virus diseases.

On the fruits, the effects are usually mottling and russeting, such as is shown by cucumbers and chillies, and the reduction of seed production and the dwarfing and distortion of those produced. The latter type is often characteristic of the cucurbit and chillie virus diseases. Cucumbers may be so warty and deformed as to be hardly recognizable.

Tissues Invaded

Some tissues offer little resistance to the viruses and others appear to act as barriers to their invasion. Bennet (51) recently reviewed this question and came to the conclusion that the tissues most likely to be invaded by viruses are the phloem and the parenchyma. The meristematic tissue is not often invaded. A few viruses are restricted to the parenchyma and a few to the phloem, but most of them are found in both.

It is the opinion of investigators at this time that viruses cannot move through cell-walls but are restricted to streams of living protoplasm. That is, they may only move from cell to cell by way of the plasmodesma. The phloem with the sieve tubes permitting connection between the living protoplasm of the cells would appear to offer the best channel by which a virus may move from one portion of a plant to another. If this is true then it would seem reasonable to conclude that the plasmodesmata are universally distributed throughout the phloem and parenchyma tissues.

Whereas bacterial diseases of plants are largely seed transmitted, virus diseases do not appear at this time to be scattered in this way. As stated earlier, the viruses are unable to enter the embryonic tissues and the fact that the embryos are largely composed of this tissue would indicate that seed dissemination would be a minor factor in the field. Nelson found that the transmission of bean mosaic by way of the seed was very irregular. He studied the vascular connection between the individual beans in the pod and the pedicel. He found that the bundles divided before entering the pod and one-half went to one seed and the other half would go to another seed. Seeds one and three would be connected to the same vascular bundle and seeds two and four to another. Nelson found the number of seeds in a pod that became infected to be very irregular. There was evidence to indicate that the vascular tissues did play a part but it was the phloem and not the xylem elements. The question would be asked at once, "How could seed infection take place through the vascular tissue when there is no connection between the mother plant and the embryo?" Nelson states that it has been shown that very little seed infection does take place after flowering. Before that time the embryonic tissue which forms the seeds is not organized into the various tissues and there are probably uninterrupted protoplasmic connections which would make possible invasion of the primoradial seed tissue by the virus. He (Nelson) found no evidence of mosaic transmission by the pollen nor was there any evidence of transmission by rust (*Uromyces appendiculatus*) which was taken from an infected plant and caused infection on a healthy plant.

So far there is no evidence that the virus particles possess any means of locomotion. Lack of evidence may not necessarily be taken to mean that they do not have any power of movement but until we can prove it we dare not assume it and must go on the assumption that only physical and chemical forces operate. Thus it seems that the virus elements must move up and down the conducting vessels under the same impulses and in the same way as the food elements and that the rate of movement closely approximates that of sugar.

Mode of Dissemination

One of the first studies conducted on the virus disease was on the mode of dissemination and the first agents of transmission were insects. To-day insects are held responsible for the transmission of a large majority of the viruses which produce disease in plants. But there are numerous other agents of transmission also and these must be taken into account when studying the viruses. It has been suggested by a number of workers including Quanjer (632), Kunkee (357) and Johnson and Hoggan (318) that a virus classification could be based on the mode of dissemination. This will be discussed further under the section on classification.

Insects with sucking mouth parts, such as, leaf hoppers, aphids and thrips, are the most common transmission agents while the members of the order *Hemiptera*, which would include the stink bugs (*Pentatomidae*), chinch bugs (*Lygaeidae*) and the squash bugs (*Coreidae*) are of somewhat lesser importance.

Transmission by seed has already been discussed in part. As was indicated, seed transmission is not common as a means of virus dissemination. A search of the literature reveals only a few instances of virus transmission by seed. Bean mosaic, maize mosaic up to about 1%, cucumber mosaic occasionally, Petunia mosaic in Petunia seed and tobacco seed and redclover mosaic have been recorded.

Transmission of pollen borne mosaic has not been proved an important means of virus dissemination because of the apparent inability of the virus to live in embryonic tissue.

Transmission by mechanical means is common. In the nursery the transmission by budding and grafting is common and it was by grafting that the first transmission of the virus disease was actually accomplished. Failure to sterilize the tools used in budding and grafting has been responsible for much of this type of transmission. To a lesser extent the same thing may be said of pruning. A virus may be latent in the buds or branches which are used in the propagation nurseries and in this way introduced onto new plants. It has been found that the virus of tobacco may live over in the dead tissues for some time and in this way workers may accidently or unconsciously scatter it. Jones and Burnett (324) stated that the mosaic common to tobacco and tomatoes may be spread among tomatoes by workmen using tobacco from diseased plants. This indicates that the virus is not killed by drying and, in the case of smoking tobacco, even by high temperatures. In the fields, the cultivating tools and animals and man himself are agents of dissemination. Birds may also scatter such diseases but are probably of minor importance.

Classification of Viruses

The arrangement of the viruses in an orderly, systematic scheme has been one of the most difficult tasks facing the virus worker. Several attempts have been made but the lack of knowledge has made the task extremely difficult and all attempts have been subjected to a great deal of criticism. No doubt any future attempt will meet the same fate until our knowledge about the viruses is sufficient to make a satisfactory classification possible.

One of the first to attempt to classify the viruses was James Johnson (318) who arranged the then known potato viruses into a key based on the chemical and physical properties. But too little was known about them and the key was unsatisfactory. In 1935, Johnson and Hoggan (319) used the mode of transmission as the basis for classification. They set up a dichotomous key using two main groups which are distinguished by the mode of transmission. A brief outline of the key is given below. This key differs from Johnson's first attempt in that it contains all of the viruses.

Dichotomous Key of the Plant Viruses

A_1 Transmissible by sucking insects.
  B_1 Not transmissible mechanically by plant extract.
    C_1 Not transmissible by aphids.
      D_1 Transmissible by leaf hoppers.
          In this group are listed the peach, sugar beet, corn, sugarcane and rice viruses.
      D_2 Not transmissible by leaf hoppers.
        E_1 Transmissible by thrips.
          Pineapple virus.
        E_2 Not transmissible by thrips.
          F_1 Transmissible by white fly.
            Tobacco and cotton viruses.
          F_2 Not transmissible by white fly.
    C_2 Transmissible by aphids.
      D_1 *Rosaceae* susceptible.
          Raspberry , blackberry and strawberry viruses.
      D_2 *Rosaceae* not susceptible
          Potato leaf roll, potato yellow dwarf, banana bunchy top, plant resette and pea mosaic viruses.
  B_2 Transmissible mechanically by plant extract.
    C_1 Longevity in vitro less than 7 days at 22° C.
          Tomato spotted wilt, potato mild

mosaic, cucumber mosaic and onion yellow dwarf.

C_2 Longevity in vitro 7 days or more at 22° C.

D_1 Thermal death point below 80° C.

Alfalfa virus and ordinary tobacco virus.

A_2 Not transmissible by sucking insects.

B_1 Transmitted mechanically by plant extract.

Potato ring spot and tobacco ring spot viruses.

B_2 Not transmitted mechanically by plant extract.

C_1 Transmission by grafting.

Potato witches broom and peach rosette viruses.

C_2 Not transmissible by grafting.

Potato giant hill and cotton crazy top viruses.

This is only an outline of the key given as an example of what may be used for such a classification. In this key the main characteristics are: (1) the mode of transmission, (2) natural or differential hosts (3) longevity in vitro and (4) the thermal death point.

In 1931 Quanjer (632) presented an attempt at a classification of the potato viruses in which he introduced the word "viroses". He criticized the classification of Johnson (318) because he had omitted to make use of the ability of the virus to produce disease as a character in the classification. He proposed a classification based upon the characters appearing in the outline below.

Outline of Virus Classification by Quanjer

A. Indirect methods.

1. Symptomology.
2. Morbid anatomy and physiology of the host.

3. Determination of the host range.
4. Determination of the modes of transmission and the relation between vectors and viruses.
5. Determination of the effect of environment on the diseased host plant.

B. Methods not yet classified as direct or indirect.

6. Cytology (X-bodies)

C. Direct methods (Property methods according to Johnson.

7. Cultivation of viruses and determination of their physical and chemical characteristics.

Quanjer suggests the use of certain terms (such as the word "viroses" mentioned above) which he believes would avoid confusion. He suggests such words as: anecrotic mosaics, phloem necrosis mosaics, etc. It might be repeated here that the use of the word "viroses" has not become popular and is not generally used.

In 1935 Kunkel (357) reviewed the status of the virus classification up to that date and came to the conclusion that before a satisfactory classification could be made, new methods of studying the viruses themselves were necessary. He believed that the hope of a satisfactory classification of the viruses lies in the use of serological methods. He reasoned that by use of serological tests, similar to those used with animals, it would be possible to classify the plant viruses.

In the same year that Quanjer made his observations and conclusions, Chester (104) presented evidence to show that certain of the plant viruses are distinct serological entities. That is, when they are injected into the body of an animal (as for example, the rabbit) antibodies are formed in the blood and when serum from this animal is mixed with some of the same virus in solution there is a precipitation and clearing of the liquid. This precipitation and clearing will occur only with the same virus or a very closely related one.

Chester presented evidence to show that by such a test he had been able to establish as distinct entities tobacco mosaic, potato latent mosaic, potato mild mosaic, potato aucuba mosaic and tobacco ring spot mosaic. At the same time he stated that the antigenic property of the virus in question is definitely associated with the living tissue and ceases to be at the death of the virus.

In 1939 Bennett (52) suggested five characters which he believed were of greatest value in classifying the plant viruses.

Given in the order which he suggested, they are:

1. Type of symptoms produced on different species and varieties of susceptible plants.
2. Morphological and cytological disturbances produced.
3. Relation of insect vectors to virus transmission.
4. Antigenic reaction in animals and plants.
5. Chemical and physical properties of the viruses themselves.

Thus we see in this suggestion the inclusion of all of the major characters which have been used in the previous classification. However, he does nothing more than suggest and offers no framework upon which to hang such a scheme. Perhaps he has been wise for in so doing he has escaped much of the criticism that was sure to follow the offering of any classification scheme at this time when the knowledge of the viruses is in such a state of flux.

At the same time and in fact, the same issue of the periodical in which the article of Bennett (52) appeared, was an article by Holmes (288) in which he offered a complete new outline for virus classification. The outline is given briefly here but the student is directed to his Handbook of Phytopathogenic Viruses, for a more detailed study.

Proposed Classification of Viruses by Holmes

Division 1. PHYTOPHAGI Viruses parasitic on plants, phytophages.

Class 1. *Schizophytophagi* Viruses parasitic on schizophytes; schizophytophages.

Family 1. *Phagaceae* Viruses parasitic on bacteria; bacteriophages.

Genus *Phagus.*

(Under this genus are six species).

Class II. *Spermatophytophagi* Viruses parasitic on flowering plants; spermatophytophagages.

Family 1. *Chlorogenaceae* Yellows group; viruses causing diseases mostly characterized by stimulation of normally dormant buds to form witches brooms, by chlorosis without spotting, or by both brooming and chlorosis. Invaded parts usually abnormally errect. Vectors typically leaf hoppers (*Jassidae*).

Genus *Chlorogenus.*

(Under this genus eight species are given).

Family 2. *Marmoraceae* Mosaic group; viruses causing diseases usually characterized by persistent chlorotic or necrotic spotting, and often by mottling; no stimulation of normally dormant buds; usually no recovery; if recovery occurs, no immunity from reinfection. Vectors typically aphids (*Aphididae*), sometimes thrips (*Thysanoptera*), or leaf hoppers (*Jassidae*).

Genus *Marmor* (from L. marmor n., a mottled substance, marble).

(Under this genus are fourteen species and thirteen varieties).

Family 3. *Annulaceae* Ringspot group; viruses causing diseases characterized by necrotic or chlorotic spotting with concentric-ring lesions; eventual recovery with non-sterile immunity. No insect vectors known.

Genus *Annulus.*

(Under this genus are two species and three varieties).

Family 4. *Gallaceae* Fiji-disease group; viruses causing diseases characterized by proliferation of normally inactive tissues; chlorotic and necrotic mottling absent systemic chlorosis, or witches'-broom, if formed, not of spindly shoots.

Genus *Galla.*

(Under this genus only one species, Fiji-disease virus).

Family 5. *Acrogenaceae* Spindle-tuber group; represented by a virus causing disease characterized by abnormal growth habit, without chlorotic or necrotic mottling, systemic chlorosis, or witches'-broom formation.

Genus *Acrogenus.*

(Under this genus only one species, potato spindle-tuber virus).

Family 6. *Rugaceae* Leaf-curl group; viruses causing disease characterized by arrested development of invaded leaf tissues, resulting in leaf curl, enations and other deformities. Vectors typically whiteflies (*Aleyrodidae*).

Genus *Ruga* (from L. *ruga* f., a wrinkle).

(Under this genus are three species).

Division II. ZOOPHAGI Viruses parasitic on animals.
  Class I. *Arthoropodophagi* Viruses parasitic on arthropods.
  Class II. *Chordatophagi* Viruses parasitic on chordates.

Here we see a new classification designed to include only the viruses, with two new divisions, and their corresponding classes, families and genera which, with a simple extension, will include the viruses which may be added to the known list in the future. One question that will immediately arise is where in the present classification scheme of the plant kingdom will this section fit? There is, of course, the vague possibility that viruses may not be plants but it does not seem at this time that we are in a position to place them anywhere else. At this time it seems that we must put them below the bacteria and thus they become the most primitive of the plant kingdom in our classification.

The student should read A Handbook of Phytopathogenic Viruses by Holmes (288) and at the same time A Textbook of Plant Virus Diseases by Smith (717), Murphy (434) criticized the work of Smith because he designated the viruses by name which he (Murphy) contends cannot be done at this time because of our lack of knowledge.

Recently Fawcett (213) proposed a classification for the plant viruses in which we add the stem "vir" for virus (Latin neuter) to the Latin genetive of the genus of the host in which the virus was first discovered and recognized, dropping any final consonants that occur in this genetive. Thus for the citrus virus the name *Citrivir* would be applied. For the virus causing psorosis he would propose the name *Citrivir psorosis.*

In 1940, Valleau (864) proposed a classification of the tobacco viruses based on the serological relationships. This agrees with the proposals of Kunkel (357) and

Chester (104). He believes that the serological method will prove the best when it is clearly demonstrated that only one virus was used as antigen. He does not agree with Kunkel in the value of the immunity reaction. He is of the opinion that there is danger of confusion when various strains of viruses are used in inoculation trials to test for immunity because of the difficulty of distinguishing between symptoms.

From the discussion so far it is clear that we have no accepted classification for viruses at this time.

CHAPTER III

THE DISSEMINATION OF PLANT DISEASES

The question of plant disease dissemination is one that the plant pathologist has to study in detail if he is to know the problems of control and especially if he is to accomplish his objectives. In the following pages will be discussed the spread of diseases by insects, weeds and air. Each of these is an important channel for the spread of plant disease pathogens.

INSECTS AND THE SPREADING OF PLANT DISEASE

The list of known insect vectors of disease is long. In the case of certain plant and animal diseases there is no other way of transmission from one host to another. For example, the transmission of malaria is dependent upon the mosquito. The Texas cattle fever organism must be carried by the cattle tick. Viruses of plants, while perhaps not absolutely dependent upon the sucking or chewing insects for transmission, nevertheless owe most of their spread to the insect carriers.

Leach (366) gives six ways in which insects may influence the spread of disease.

1. Direct production of toxic substance.
2. Dissemination of the pathogen.
3. Inoculation of the suscept with the pathogen.
4. Ingression of the pathogen into the suscept.
5. Invasion of the suscept by the pathogen.
6. Preservation of the pathogen.

In the first case the insect may produce a toxic substance that may cause a diseased condition even though no pathogen is actually present. The question of the dissemination of the pathogen should need no comment. In the case of inoculation of the pathogen it is carried from one place to another. The transfer is to a place where it may cause infection and disease. Dissemination and inoculation may be performed in the same act but not necessarily so. Spores of many fungi are carried on the feet of insects but infection does not always follow.

Ingression is gaining entrance to a place where growth may continue. It may be that the agent of ingression has little, if anything, to do with the dissemination of the fungi. Strictly speaking, wound parasites are not usually carried by the insects that make the wounds. Anthracnose of papaya may start on the fruit but the insect that makes the wound for the entrance of the fungus may not have carried the spores at all. There is greater evidence that the wood-boring insects may carry the spores of wood-rooting fungi as they move about in the tunnels they have carved out in the trees.

Plant pathogens find it difficult to survive through all the seasons in all parts of the world. To be of a serious nature they must find a means of preservation. Insects may be the means of carrying such plant disease agents as viruses and bacteria over the period of the year when the host is dormant. The sudden new appearance of a disease may have been due to the introduction into a community of the insect vector or of the finding of a new relationship between insect and the plant disease organism. It is suspected that the Dutch Elm disease, caused by *Ceratostomella ulmi*, was introduced on logs of elm taken to the United States for veneering purposes but there it was spread by insects which become associated with it after its introduction. The Scolytid bark beetle makes the wounds for the fungus to enter. These beetles, carried from one place to another, carry the fungus along with them. The losses due to the elm diseases have run into many thousands of dollars and thousands of acres of fine timber have been destroyed. Quarantine and eradication have helped but the real answer appears to be in a hybrid resistant elm, the "Christian Buisman", which was named for the originator after her untimely death.

There are numerous biological relationships between fungi and insects that play a very vital part in the life of the fungus. Some of these have existed for a long time. In the case of Coprinus, the inky cap fungus, insects bring about the fusion of the basidiopsores. In black stem rust of wheat, flies, or other insects bring about the union of pycnospores and receptive hyphae resulting in the production of aecia and aecidiospores.

Ergot of rye (*Claviceps purpurea*) is disseminated by flies. In the case of blue stain of conifer trees, (*Ceratostomella sp.*), it has been shown that dissemination is by a bark beetle. The fungus possesses perithecia with long neck which protrude up in the floor of the burroughs and as the beetle crawls along it picks up the spores thus distributing them over the area it has worked. An internal rot of figs known as "endosepsis" is caused by *Fusarium moniliforme* var. *fici*, and carried by the fig wasp, *Blastophaga psenes* L. Perennial canker of apples, caused by *Gloeosporium perennans*, it being really not perennial, as the name indicates, but each year starts anew from the base of the old canker which is injured by the wooly apple aphis.

Sooty mould, caused by species of *Meliola* and *Capnodium*, is not a real parasitic disease, but the result of the activity of saprophytes. They, however, reduce the sunlight and thus interfere with the proper metabolism of the plant. The fungi grow on the secretion of certain of the plant lice. The secretion, known as honey dew, is sweet and thus offers a very excellent medium for the fungus. Ants carry the aphids about and they in turn secrete the honey dew upon which the fungi live. Thus there is a somewhat complicated relationship between the insects and the fungi. The potato flea beetle (*Epithris cucumeris*) carries potato scab (*Actinomyces scabies*) (Thaxter) Gussow. Cabbage black leg (*Phoma lingam* (Tode) Dezmaz) is carried by the cabbage maggot (*Hylemyia brassicae* Bouche) Red rot of sugarcane (*Colletotrichum falcatum* Went) is associated with the cane moth borer (*Diatroea saccharalis* F). There are many instances of the relationship between insects and the fungi which cause plant disease but these will be enough to give an idea of the range and type.

Virus Diseases

Leach (366) gives five orders in which virus diseases vectors are included. These are the *Orthoptera*, *Thysanoptera*, *Homoptera*, *Hemiptera* and *Coleoptera.* He considers that the *Orthoptera* are only mechnical agents of transmission. The *Thysanoptera* have been shown to be the vectors of spotted wilt of tomato. The *Homoptera* are the most important of the virus carrying insects. This order includes the aphids, leaf hoppers, white flies and scale insects. Many of them are virus carriers. Some of them, as for example, *Myzus persicae* (Sulz) the green peach aphid, may carry a number of different viruses about. Many of the insects, however, are specific in regard to the virus they carry about.

Leaf hoppers also play an important rôle in the transmission of virus diseases. Among the diseases they are reported to transmit are, the Fiji disease of sugarcane and the curly top of sugar beets. The true bugs, *Hemiptera*, are also important as virus disease transmitters. Some of them transmit potato virus diseases such as spindle tuber and potato blight. Some of them are evidently responsible for the transmission of the virus disease of bhindi. Beetles (*Coleoptera*) are mainly chewing insects and thus probably play only a mechanical rôle in the dissemination of virus diseases.

The question of the length of time the virus remains alive in the insect body is important from the point of view of the plant pathologist. This has to do with the dissemination and thus enters into any control program which may be devised. Whether the virus can increase inside the body of the insect is a question that has received a great deal of attention. So far the evidence does not positively support the idea that the virus may increase inside the insect body. Where the virus has been known to survive for a long time within the insect body it has been assumed that multiplication took place. However, this is not positively shown.

Numerous experiments have been conducted showing that the virus is closely related to the insect body and that anything that might influence it would be likely to also influence the virus. Kunkel (359) was able to show that the aster yellows virus could be inactivated within the body of the insect by heating the insect to a temperature of 31-32 degrees C. for a sufficiently long time. A shorter time did not inactivate the virus. The temperature in this case did not appear to have any effect upon the insect. Transmission of the virus by way of the egg has been shown in the case of a virus disease of rice. But this is not to be construed as meaning that all of the viruses are carried within an insect body, as many are not.

The time required for the insect to become viruliferous may be as long as one minute or it may be a number of days. Kunkel found that 10 days was required for the aster yellows virus to become effective in the body of the vector. Why this delay in the time of insect transmission is not clear but it is rather generally held that the time is required for the particles of virus to be absorbed into the insect blood stream and then travel to the salivary glands from whence it may be injected into a host plant.

In certain cases there appears to be a specificity for a certain virus on the part of the vector. Johnson and Hoggan (319) show apparent specificity of viruses when they constructed the table for the classification of the plant viruses. On the other hand yellow dwarf of onions can be transmitted by some 50 different species of aphids. In 1934 Smith (717) classified the plant viruses into four more or less distinct groups according to the insect carrier. He classed them as (a) the mosaic group carried by aphids; (b) the yellow group carried by leaf hoppers; (c) the ring spot type carried by thrips and (d) the group causing leaf and stem distortion carried by white flies. Just what it is that causes the specificity of an insect for a particular virus is not known but is generally believed to be due to the permeability of the intestinal walls to the virus particle.

Leach (366) lists 24 virus diseases of important economic plants that are transmitted by insects. Host plants listed are potato, tobacco, onion, cucumber, celery, sugar beet, sugarcane, aster, maize, rice, peach, cranberry, tomato, pineapple, cotton, casava, black current, wheat. The insect vectors are aphids, leaf hoppers, thrips, mites, bugs, etc. A complete list of the viruses and vectors is outside the limits of this book.

Transmission of fungus, bacterial and virus diseases is not confined to insects alone. Nematodes and other small animals of the soil have been shown to be vectors. The nematode, *Heterodera marioni*, which is so common in India, is no doubt a carrier of some of the fungi associated with the root rots. The cereal nematode, *Tylenchus tritici*, (Steinbuch) Bastian, has been shown to carry the spores of certain leaf-spotting fungi. Cotton seedling damping off and root rot of sugarcane have been shown to be transmitted by nematodes. Earth worms have been shown to be able to transmit some of the soil-borne fungi such as club root of cabbage.

Weeds as Carriers of Plant Diseases

The roll of weeds in plant disease carriers is an important one. There are many plant diseases to-day that are found on weed hosts and it appears that the studies so far have been very incomplete. This is especially true of the soil-borne organisms such as *Sclerotium rolfsii, Rhizoctonia solani,* and *Fusarium* species. Butler and Bisby mention a number of instances of fungi causing loss to economic plants that are found on weeds. For example, *Piricularia oryzae* (96) has been reported on species of *Panicum, Setaria* and *Paspalum. Colletotrichum capsici* is found on *Vigna catjang, Solanum xanthocarpum, Datura fastuosa, Hibiscus esculentus* and others. Some of the rusts, such as *Puccinia himalensis* (*P. coronata*), *P. graminis* and *P. glumarum* have been reported on grasses that are classed as weeds when found among other crops. However, the latter are not considered as important in the spread of the rusts.

Neocosmospora vasinfecta has been isolated from the roots of a wide range of host plants, both monocotyledon and dicotyledom. The powdery mildews are extremely cosmopolitan in their choice of host plants and such powdery mildews as *Erysiphe cichoracearum* and *E. polygoni* are apparently little handicapped by the absence of any one host plant. Butler and Bisby (96) report *Sclerotinia sclerotiorum* on some 23 different host plants and since then Mundkur has reported it on *Hibiscus.* The same authors record *Pythium aphanidermatum* on some 21 different hosts. The *Phytophthora* species are also widespread. *P. arecae* is recorded on 12 different hosts and *P. Palmivore* has been recorded on 7. *Cystopus,* the white rust, is also wide spread.

The fact that pathogenic fungi have been isolated from so many weed plants and that artificial infection of weeds has been accomplished by numerous workers, indicate that they are a potential threat as hosts and that they will have to be considered in any control program.

Virus diseases are known to be widely spread among the weed hosts. See under Solanum Virus I. In many cases the control measures for viruses definitely recommend the destruction of the weed hosts about the farm or cultivated area.

Recently Wilson (932) has given an excellent review of the literature on the control of noxious plants and it will be worth the time of the student to look up this article and read it. In the article he suggests a wide variety of control measures which include tillage, cropping, and chemicals. Among the chemicals suggested are sulphuric acid, sulphates, Sinox (sodium dinitro-orthocresylate), Arsenicals, chlorates, and others. There are numerous so-called selective weed killers on the market under various trade names. Each is effective against some types of weeds and under certain circumstances. Sinox is one that is commonly used in the United States.

The machinery for application of weed killers on large acreages is expensive and difficult to secure at this time. Small units are becoming available for use on small areas and these are not so expensive and no doubt will soon become available for use in India.

The present method of removing weeds is both expensive and destructive to the crop plants. The selective weed killers do not harm the crop plants and the damage to the roots caused by the removal of weeds is avoided.

Plant Disease Through the Air

During recent years the attention of the biologist and plant pathologist has been attracted to the air as a possible means of dissemination of plant and animal disease. In India, Dr. K. C. Mehta of the Rust Research Laboratory Flowerdale, Simla, has been among the leaders who have pointed out the possibility of the air as a conveyor of rust spores. In the United States, Charles Lindberg exposed vaseline covered slides up to 12,000 feet in the air and found spores of fungi caught on them. Durham (203) found that when he exposed 2,000 vaseline-coated slides in 40 localities in the United

States over a period of four years, he found the number of *Alternaria* spores per cubic yard of air, for a 24-hour period, to be over 25. Prince and Morrow (628) found the spore number per cubic yard for a 24-hour period for such moulds as *Monilia, Aspergillus, Penicillium, Trichoderma, Homodendrum, Helminthosporium* and *Cladosporium*, to be in the neighbourhood of 2,688. These were air borne from the swampy lands to the coastline of Texas. But most of the dissemination studies are in terms of local areas.

Fungi produce spores in enormous numbers and thus offer great opportunity for air dissemination. It has been calculated that a fruiting body of *Daedalia confragosa*, 2 square inches in size will produce 682,000,000 spores (735). A large fruit body of *Fomes applanatus*, will produce 5, 460, 000, 000, 000 spores and could discharge 30,000,000,000 a day for six months. *Sclerotinia sclerotiorum* produces 31,000,000,000 ascospores. A single wheat kernel which has been converted into smut (*Tilletia tritici*) may contain 12,000,000 spores. Stakman and Christensen (735) estimated that an acre of wheat containing 1 per cent of smutted kernels would contain 5,000,000,000,000 spores. On one acre on slightly rusted wheat they estimate 10,000,000,000,000 spores. What would be the estimate on heavily-rusted wheat on the same area?

From a study of the fungi it is evident that nature prefers many small spores to a few large ones. The utilization of spore-producing space by the fungi is remarkable. The basidiomycetes are among the most efficient. But one must marvel at the spore wastage. Only a small portion of the spores ever germinate and of the ones that do germinate only a small portion ever produce infection on a host plant.

Methods of spore release are varied and often ingenious. They vary from the spores which, like those of the downy mildews, must await the decay of the host tissue before release to those of the fungus *Pilobolus* which fires them from a sporangiophore which is phototropic in its reaction. Many of the *Ascomycetes* fire their ascospores into the air for some distance but depend upon the wind to carry them on to another host. In the case of *Empusa,* which is one of the entomophagous fungi, the spore is not only fired from the sporangium but it also carries enough reserve gas so that, in case it does not strike on a fly, it may set up a secondary base and be fired again. But by far the greater number of the plant pathogenic fungi are distributed by the wind or insects.

The spore size may vary from 5 microns to 300 microns. Pycnospores and ascospores are small whereas, some of the conidia of the *Imperfect Fungi* are over 200 microns. Some may even reach 300 microns in length. Small spores may travel many miles from the source of origin. They were found in the spore trap of the Explorer II which went to 36,000 feet for the collection. Carried upwards spores may be carried for long distances before coming down. Stakman and Christensen (735) gave the distance, a spore would travel while falling one mile within a wind of 20 miles an hour, as 2,900 miles. The estimation was based on laboratory tests.

A knowledge of the true rate of spore discharge is of considerable importance. Work on the apple scab fungus (*Venturia inequalis*) in the United States, has yielded valuable results on the time and rate of discharge of the ascospores. Data collected show that spores are discharged after rains but more or less independent of temperature. How far the ascospores of apple scab will travel after discharge is not clear but the leaf infections that occur indicate that the spores are somewhat resistant to drying. The knowledge of the time of the spore discharge is used in preparing the spore schedule.

Wind dissemination of the spores of the late blight of potatoes is very definitely known and it has been traced as much as 600 feet from old refuse piles which were infected. Precise information is difficult to obtain because the epiphytotics are usually sporadic and thus give little opportunity for comparison of one year with the next. Weston (928) reported that *Sclerospora* sp. conidia are disseminated for considerable distances in the Philippines even in still weather. Spores have been caught as much as 80 feet from the infected plants. They were able to trace the infections for several miles from the original source by following down the prevailing wind direction. The spread being from field to field with occasional jumps from island to island.

The length of life of the fungus spore will have considerable effect upon the distance it may travel and produce infection. As already indicated, *Sclerospora* conidia may travel a long distance and retain the power of producing infection. The basidiospores of the cedar-apple rust (*Gymnosporangium juniperi— virginianae* are very short-lived and cannot cause infections for great distance from the cedar trees. White pine blister rust (*Cronartium ribicola*) is also limited by being short-lived. It is also limited by the fact the alternate host must function between the place where the aecidiospores are produced and the next white pine to become infected. Species of the wild goose berry (*Ribes*) are the alternate hosts. If these can be eradicated for a distance of about 900 feet from the pine host infection of the white pine will not take place.

Few diseases are spread over wide areas in a single season. Smuts, such as *Ustilago tritici* and *Ustilago nuda*, while they are well adapted for wind dissemination, cause few infections at any great distance from the infection centres. All the factors against the effective dissemination of smuts are not known but it is considered that differences in time of spore formation and host susceptibility may be a major factor. Also, secondary infections do not take place the same season as the primary ones and this would reduce the rate of spread. In the case of flag smut (*Urocystis tritici*) spores apparently travel for long distances with the wind but infections are confined to small areas for a considerable time. *Ustilago zeae* and *Sorosporium reilianum* are both readily disseminated by wind. Although seed treatment of corn smut is not effective, there is little real evidence that the infections come from wind-borne spores.

Long-distance dissemination is most conclusive in the case of such diseases as the downy mildews, powdery mildews and the rusts. From the wheat area in Canada to the southern end of the belt in Mexico is a distance of some 2,500 miles in which wheat is grown without a break. Susceptible plants are available for the rust in some part of the area throughout the entire period. Thus, even without the barberry, the rust inoculum is available and there are susceptible hosts. Uredospores, which are produced in enormous numbers, maintain their vitality for months under moderate temperatures.

Mehta (453) has stated that the uredospores of stem rust and leaf rust of wheat cannot live over the hot season on the plains of India and therefore must come from the hills each season to start the infections. He has stated that it is possible to follow the infection from the hills to the plains by progressive stages that would represent the jumps which wind-borne spores might be expected to make. Stakman and Christensen (735) review the findings of the 1923 season in the United States. They state that uredospores remained alive in the southern part of Texas during the winter of 1922-23 and, from the results of spore trappings with vaselined slides in 38 stations distributed throughout the states of Texas, and north to Minnesota, it was possible to show that it was the wind-borne spores that was the source of the inoculum to start the infections. This was also confirmed by the distribution of the physiologic races. From the observations and data collected it was considered as reasonably certain that wind-borne spores of stem rust, *Puccinia graminis tritici,* were distributed from Mexico to Canada in a single season. Other years were not so conclusive but the evidence is all in favour of the wind-borne spores as the source of the season's infections.

In 1934 a southerly wind carried spores of stem rust south in October and created an epiphytotic in southern Texas thus laying a foundation for the 1935 season. As a result of this heavy infection in 1934 the wind load of uredospores going north in 1935 was enormous (735). By June 15, slides exposed in the state of Nebraska caught 4800 spores per square foot in a twenty-four hour period. By June 22 as many as 15,000 per square foot were caught at Fargo, North Dakota. By June 25 the spore count had risen to 92,600 per square foot per 24 hours and this at a distance of some 2,250 miles from the original source of the epiphytotic.

How great a part the barberry plays in the epiphytotics from season to season is not clear. Aecidiospores have been caught at 7,000 feet and they retain their vitality for a long time. The United States and Canada may be divided into zones which are limited by wind currents and natural barriers as well as physiologic forms. High mountains and water barriers limit some of the forms to one area to such an extent that physiologic forms of one area are not known in another. How far spores may travel in the air is not known but spores have been collected at long distances from the source. Whether they can travel the long distances attributed to them and still cause infection is not known.

CHAPTER IV

AGENTS OF PLANT DISEASE CONTROL

In the control of plant disease there are a number of agents that may be used. Perhaps the first control agent that the plant pathologist would think of would be the chemical dusts and sprays. These are among the oldest and best known. More recently other factors have entered into the realm of plant disease control such as the biological agents. Numerous articles regarding the antibotic agents and the control of disease have appeared in recent literature. Perhaps the first of these is the bacteriophage and only within the last decade have we heard much of the toxic substances which are a by-product of the living animal and plant cell. The use of these is just beginning and only the future can predict what may come from the many experimental efforts being made. Before closing this portion of the chapter a word will be said about the trend of development in the production of fungicides.

One of the very important agents of disease control is that of the quarantine. Plant pathologists are too often likely to say that the job of quarantine is in the realm of the Government and thus not take an active part in it. But the Government is composed of the citizens of the country and, as a citizen, each one of us is responsible for the working of the Government. Therefore a knowledge of the quarantine laws is essential and, with the knowledge, cooperation to make them effective.

Chemical Agents in Plant Disease Control

Disease control consists of either killing the fungus parasite or in producing conditions that are so unfavourable for its growth that it cannot attack the host plant. Under the first condition the fungus is attacked directly by use of chemicals or by biological agents. Under chemical agents would be listed such elements as copper, sulpher, formaldehyde, etc. Where seedborne fungi or root parasites are concerned such elements and compound as the mercury compounds, sulphuric acid, creosote, lye, carbolic acid, borax and others, are used. Recently a new series of compounds are being sold on the world's markets under a variety of trade names. On the markets of the United States one finds such compounds as Fermate (ferric dimethyl dithiocarbamate; Semesan (chlorophenyl mercury) Arasan (tetramethyl thiuram disulfide) of the E. I. du Pont de Memours Co.; Spergon (tetrachloropara benzoquinone) of the United States Rubber Company and many others which are not yet fully tested.

Liquid sprays were first used to control fungi on crop plants. From the beginning to the present date copper and sulphur sprays have been the most prominent of the liquid sprays to be used in the control of fungi. More recently other elements have been used successfully and it appears that we may expect more of these non-copper or sulphur sprays to appear. Fermate mentioned above, does not contain either copper or sulphur.

Perhaps the factor which did most to stimulate the development of sprays, dusts and their use, has been the invention and improvement of spray machines and equipment. Without the improvement and introduction of improved spray machines it is doubtful if the advance in spray materials would have been made. It is a far cry from the time when the first spray were sprinkled on the plants with a broom or brush to the huge power units which may be seen in the larger orchards of the world to-day. With the introduction of the improved machines it was possible to give each new formula a fair and complete trial. Complete and efficient coverage of the plants became the watchword and spraying and dusting became a science.

There are a number of factors which must enter into the consideration of a spraying or dusting schedule. Among the more important are: (1) the area involved, (2) type of disease concerned, (3) number of applications of spray or dust needed and (4) availability of materials such as labour, water, electric power, etc. For small areas the bucket, or wheel barrow, type of hand spray or the small type hand duster may be sufficient. If field crops or orchards are involved then some form of power machine will be needed for efficient and effective work. Many types of power machinery are now available so that the farmer has a wider range from which to choose and it is possible to secure the proper machine for any condition likely to be found. For the small orchard or field there is the 50 to 100 gallons outfit which may be powered by traction or by motor. It is usually hauled about by animals. For the large orchardist or grower there is the motor-driven outfit of 200 to 250 gallons capacity. For field crops it may be satisfactory to have the power come from traction but for orchards it had better be motor driven. For the yard or garden there are many types of small hand sprayers and dusters available. The catalogues of most of the larger reliable seed houses will contain illustrations of hand sprayers and dusters. It will probably be necessary to write for literature on the larger outfit.

Sprays

Copper

The use of copper for spraying was brought about accidentally. In the southern part of France the vine growers were bothered by people stealing the grapes along the roadsides. They used to put verdigris (a greenish coloured substance formed by the action of acetic acid on copper. It is poisonous) on the vines to prevent stealing of the grapes. At one time verdigris was not available and in place of the acetic acid-copper preparation the growers substituted copper and lime. Professor Millardet, of the University of Bordeaux, chanced to pass that way and noted that the plants having the copper-lime mixture on them were bright and green whereas the plants not so treated were all brown from the mildew. He immediately asked for the story of the treatment and upon going back to the University began to experiment with copper and lime with the result that bordeaux mixture was discovered. Bordeaux mixture owes its protective power to prevent germination of spores of fungi or to arrest the growth of those that do germinate. In this way infection is reduced.

Bordeaux mixture is prepared in a great many different ways. The concentration of copper varies from 1 part copper sulphate to 4 parts lime to 50 gallons water to 6 parts copper sulphate, 6 parts lime in 50 gallons of water. The most common formula for field and orchard is the 4-4-50. The copper sulphate-lime-water are always written in that order. Preparation is an exacting process and must be done carefully.

The required amount of copper sulphate is dissolved in 4-5 gallons of water and the required amount of lime is slaked in another 4-5 gallons of water. When the lime water is cold it is strained into a tank and enough water added to make up to 45 gallons. To this is added, with vigorous stirring, the copper sulphate. The preparation is then bordeaux mixture and ready to apply. It must be applied soon after making or it will deteriorate. If this is impossible then add a heaping tablespoon full of cane sugar to the spray and thoroughly agitate.

Bordeaux mixture must not be used with iron or steel equipment as they corrode badly. All parts in contact with the spray must be of some other material.

Commercial bordeaux mixture may be purchased in the market in many types of packages and under many different trade names. But when purchasing commercial material be sure that the formula is on the package. It should be something as follows:

Active ingredients	Copper 2.41 per cent
Inert material	Lime 97.59 per cent

There are many advantages to the commercial bordeaux mixture. No stock solutions required and thus the messiness usually associated with the preparation of home made sprays is eliminated. The commercially prepared mixtures are likely to be more uniform than home-made sprays. Greater uniformity results in better coverage and thus better disease control.

If the form of the copper is given on the package it is possible to determine the amount of copper sulphate by multiplying the amount given for the active agent by a factor. For example, if the copper is in the form of "copper", or metalic copper, multiply by 3.93 to determine the amount of copper sulphate. If the copper is stated as Copper oxide (CuO) then multiply by 3.14 and if it is given as copper hydroxide ($CuOH$) then multiply by 2.56.

Bordeaux mixture has the disadvantage of burning tender foliage. Burgundy mixture, which is a modification of bordeaux mixture, is a better preparation for tender foliage. It is often substituted for bordeaux mixture for summer spraying. The formula is as follows:

Copper sulphate	1 pound
Sodium carbonate	1½ pounds
Water	50 gallons

Bordeaux mixture will have a stain on smooth-skinned fruit which is objectionable and which necessitates washing. In attempting to avoid the stains ammoniacal copper carbonate has been tried. It is made of copper carbonate and ammonia. It leaves no stain but is less effective than burgundy mixture and more likely to injure the tender foliage.

Sulphur

The use of sulphur for spray antedates that of copper. Sulphur was first used as a fungicide by a gardener in Verseilles, France, who used it on vegetables. Lime and sulphur, as we know it to-day, was first used in Australia as a sheep dip. It was used to control ticks and continued to be used for that purpose until replaced largely by the creosote dips which came later.

In the United States it was first used for the control of San Jose scale on the fruit trees of California. About 1880 it was used for the control of peach leaf curl. A chance substitution of lime sulphur for bordeaux mixture, by Dean A. B. Cordley of Oregon State College, U. S. A. demonstrated its value as a control agent for apple scab. It gave much better control with less burning of the leaves. Following this discovery it became the standard fungicide for some fruits.

There are a number of formulas for making the sulphur spray. The self-boiled lime sulphur came first but as this is troublesome to make, other formulas came into favour and commercially prepared lime sulphur sprays began to appear on the market.

Self Boiled Lime Sulphur

This is a summer spray. Sulphur and lime are mixed together as a mechanical mixture but the heat generated by the slaking lime does cause some chemical change to take place and this apparently adds to the efficiency of the spray. The formula of lime-sulphur self boiled is as follows:

Sulphur (commercial flour)	8 lbs
Stone lime (burned lime)	8 lbs
Water	50 gallons

The burned unslaked lime is placed in a barrel, or other container that is large enough to hold it and permit stirring, water is added to start the slaking.

When the slaking is well under way the sulphur is added and the mixture is stirred vigorously. More water is added as the lime sulphur mixture appears to be drying as it must not burn. Amber coloured streaks in the mixture are composed of polysulphides and may be the cause of the burning of the foliage when applied as spray. When vigorous boiling has ceased add the rest of the water to make the required amount. It should be strained to get out the pieces of lime that do not go into solution. There is a tendency to settle rapidly and for this reason there must be some form of agitation constantly going on to produce the proper results. Self boiled lime sulphur sprays have been recommended for summer sprays and in that capacity they are superior to the bordeaux mixture for tender foliaged plants.

Wettable Sulphur

These are sulphur mixtures which may be wet with water and used as sprays. The commercial forms usually contain 45 to 50 per cent sulphur and the remainder water. They may be used with arsenicals for the duel purpose of controlling insects and fungi at the same time, as for example, apple scab and the codling moth. However, if they are to be used with arsenicals then lime must be added to prevent burning of the foliage by the sulphur. The function of the lime in this case is not clearly understood. Add 2-4 pounds of lime to each pound of arsenical.

Dry-Mix Lime Sulphur

Sulphur lime calcium caseinate, mixtures are the most popular of the commercially prepared forms. The dry ingredients may be weighed into packages sufficient for the capacity of the spray tank and used at any time. The dry-mix lime sulphur may be substituted for the self-boiled lime sulphur in the summer spray schedule. The formula for the dry-mix lime sulphur is as follows:

Sulphur (Commercial flour or dusting)	8 pounds

Hydrated lime (finely ground finishing).	4 pounds
Calcium caseinate or powdered skim milk	½ pound
Water	50 gallons

There are a number of dry lime sulphur forms on the market. Most of these contain the dehydrated polysulphides of calcium and are not a substitute for the dry-mix lime sulphur.

Other Fungicides

Hydrated lime has been used as fungicide. Its value appears mostly as a repellant when used alone. However, when freshly hydrated lime has been used alone it has given some control of apple scab and apple blotch in the United States. To what the fungicidal value is due to is not clear.

Formaldehyde is used both as a liquid and as a gas. It has been used for a number of years. Formalin is a solution of the gas in water. When in contact with an organic substance it takes away oxygen. It is sold as formalin and varies from 32 to 40 per cent formaldehyde. It is used as a disinfectant more than as a spray. Seed and seed-bed disinfection being among the more important uses. When used as a disinfectant the strength varies from 2 to 2½ per cent.

Bichloride of mercury. Bichloride of mercury, or corrosive sublimate, has been used for a number of years as the standard treatment for potato scab and scurf. It has also been applied to the knives used for cutting seed potatoes and for the tools used in orchard pruning as well as sterilizing the cut surfaces of the limbs.

Cyanide of mercury has been recommended for bichloride of mercury in some cases as it is less poisonous.

Spreaders. Few spray materials will spread uniformly over the waxy surface of leaves and fruits unless there is something added to reduce the surface tension. Soaps, oil emulsion and glue have been the standard materials used for the past years. These were often difficult to manage. Glue needed hot water. Soap will not combine with all materials readily. About 1920 milk began to be used as a spreader. The calcium caseinate resulting from the milk-like mixture being the effective agent. Although the opinion is not unanimous, it is believed by a large number of the men working with spray that the casein spreader will increase the number of trees that can be covered with a given amount of material. A more uniform spread will be secured and, when arsenates are added, it does not reduce the toxicity of the arsenic.

Calcium caseinate can be purchased on the market in various forms. It is usually sold under a trade name but the product is the same. In the United States it may be sold as "Kayso", "Spreado", etc. General recommendations are add 1 to 2 pounds of the spreader to each 100 gallons of spray. The calcium caseinate should be added to the spray just before applying. Two quarts of sweet skim milk will be equal to ½ pound of calcium caseinate.

Resin is used in some cases as a sticker. It is very good when mixed with fish oil soap.

Ordinary starch paste and flour are also excellent spreaders and stickers. They exert no influence on the spray solution.

DUST

During the past two decades dust fungicides and insecticides have come into a prominent place in the program of the orchardist. There has always been an objection to the liquid spray on the grounds of the muss they cause in preparation, not to mention the equipment and time required. Dusting has a number of advantages over spraying among which the following are typical—

a. Less time required.
b. Less labour required.
c. Water not needed.
d. Less investment per acre.

e. Less motor power required.
f. Machines and material for dusting are light when compared to the same for spraying.
g. Dusting may be done when the trees are damp. It is usually best when the trees are damp. This may be a handicap in the case of spraying.

But there are certain conditions and diseases for which dusts have not proved themselves as satisfactory as sprays. The dormant dusts are not as satisfactory as the dormant sprays. Disease control has not as yet been as thoroughly worked out for the dusts as for the sprays. At the present time opinions vary widely regarding the effectiveness of the two types of fungicides. Results have varied widely under different conditions Materials for dusting are more expensive than for spraying but it is generally believed that the added expense of the dust cost is more than balanced by the saving in time and labour. The advocates of the dust fungicide argue that the time element alone is sufficient to justify the use of dusts over sprays in most cases. For example, in many cases the time which is allowed from the time of the first spray until the last is applied is very short. A good dusting machine will cover several times as much ground as the same size and power in a spray machine. The ability to get the dust on within a short time is often a very important factor. Most of the dust will be applied at the optimum time whereas it would be impossible to get all of the spray on during the optimum time and the damage will have already been done. As in the case of sprays, copper and sulphur are the two leading elements used in the preparation of dust fungicides.

Copper

Copper dusts are made from copper sulphate after all water of crystallization has been driven off. The crystals are then ground finely enough to pass a-300 mesh screen. This finely ground copper sulphate is then mixed with finely ground lime at the rate of 15 to 20 per cent copper sulphate to 80 to 85 per cent lime. This has not as yet proved effective enough to completely replace boreaux mixture in the control of such diseases as late blight of potatoes but because of the ease of application experimentation will continue until a form has been found which is equally effective.

Copper carbonate dusts are made by driving the water of crystallization from copper carbonate and grinding it as for the copper sulphate. Copper carbonate has been found very effective against smuts when used at the rate of 2 ounces to a bushel of seed.

Sulphur

Sulphur dusts, like those of copper, must be very fine (at least 300 mesh) and at least 98 per cent pure. The effectiveness of sulphur dusts is in direct proportion to the fineness of the particles. The power of adhesion is positively correlated with the fineness of the powder. Sulphur does not often cause burning. It can be combined safely with various other sprays, such as lead arsenate, calcium arsenate and lime.

Seed Treatment for the Seed-borne Fungi

The first attempt to control fungi on the seeds of plants appears to have been some three centuries ago. Some of the early plant pathologists used salt, lime, saltpeter, wood ashes and other materials. Copper sulphate was used first about 1761 but it was a century later before any definite recommendations were made for its use. The hot water treatment for loose smut was first developed by Jensen in 1887. It is still used for fungi like loose smut of wheat, which are not controlled by surface sterilization.

Formaldehyde was first recorded as a disinfectant in 1895 when it was first used in Germany. It was used in the United States in 1897. Formaldehyde and copper sulphate were the outstanding seed treatment fungicides until 1914. By that time the first mercuric compounds were being tried. Uspulun appeared on the markets in Germany. Semesan was introduced in the United States. Others rapidly came into the market. Australia was the first country to introduce dusts into the seed treatment program. The dusts immediately caught the fancy of the plant pathologists and they rapidly look the place of liquid seed treatments for the same general reasons that the dusts replaced the sprays in the control of fungus diseases in the orchard and field. They are more easily supplied, do not require water and eliminate the necessity of drying the seed. The chances of an error in the concentration of the chemical is greatly reduced as only so much of the dust will adhere to the surface of the seeds whereas the absorption of the liquid goes on as long as it is in contact with the seed. If an excess of the dust is added the excess will merely sift through the seed and lie in the bottom of the container. Also, when too much or too little dust is added to the seed, the appearance of the seed coat will tell the operator the situation. Dusts protect to some extent against further contamination by fungi and infestation by insects. They protect against soil organisms as the seed is germinating.

On the other side of the ledger the dusts are poisonous if inhaled and they usually retard the flow of seed through the drill. It has been calculated that they increase the bulk of the seed some 6-7 per cent. Within recent years a large number of fungicides have come into the market. A number of these have made use of other elements than copper, mercury or sulphur. For example, Spergon, already referred to, is a benzoquinone. Formaldehyde dust is also available and effective if kept in a tight container until needed. Its chief disadvantage is that it deteriorates rapidly.

Sulphur has appeared as a dust for seed treatment but so far does not appear as satisfactory as some of the others. Copper has proved more satisfactory. Copper carbonate has already been mentioned as a control of smut of wheat. The better grades of copper carbonate contain some 50 per cent of metalic copper made up of equal parts of copper carbonate and copper hydroxide. The better grade has proved much more effective than the cheaper grades.

Many of the chemical seed treatment compounds have appeared on the market for a short time and disappeared to be replaced by others. The good reliable ones still remain but, having been fooled by poor material once, the farmer is likely to be slower to accept another. In the United States it has become popular for the seed distributing houses, elevators etc., to treat the seed with a reliable chemical, charging only enough to cover the cost of the treatment, material and labour. They believe that in this way the increase in clean seed, they have the opportunity of handling, pays for the increase in cost of handling the seed to the grower. These practices have extended the use of the chemical seed treatment beyond that likely to have been attained if it had been left to the farmer to do.

Biological Agents

Antibiosis

The role of biological agents in the balancing of plant and animal life is as old as nature but the use of such agents in the control of plant and animal diseases on the part of man is something comparatively recent. There are two ways in which a biological agent may be useful in the control of disease. First by directly attacking the organism causing the disease and second, by secreting an antibiotic substance which indirectly reduces the activity of the disease-producing organism. Most of the knowledge of parasitism of the disease-producing organisms deal with the latter phase of the relationships.

A number of cases have been recorded in which antibiotic substances are known to be secreted by organisms which have a definitely toxic effect upon some of the disease-producing organisms. Substances like the well-known *penicillin* and *streptomycin* are examples. These two have been made famous because of their use for the control of some of the venereal diseases as well as some of the other serious diseases common in the army.

The production of antibiotic substances has been suspected for some time. Berridge (56) found that the sap of the potato appeared to contain some agglutinating principle which would agglutinate the vegetative cells of such organisms as *Bacterium tumefaciens, Bacterium solanasaprus* and *B. phtophthorus.* Most of the work on antibiotic substances has been on those produced by bacteria. In 1931 Johnson (316) reported that certain bacteria had the power to dissolve the walls of the sporidia of such fungi as *Ustilago Zeae, U. levis, U. avenae.* These include a coccus; a motile non-spore-bearing rod like bacterium; a motile, spore-bearing rod like bacillus; and a species of *Myxobacterium.* She found other bacteria producing the same type of enzymes but they were unable to affect the sporidia of the smuts.

In the same year that Johnson reported, Bamberg (74) published an article in which he states he secured evidence that at least ten different bacterial cultures possess the power to dissolve the spore walls of smut (*Ustilago zeae*Q when the cultures of bacteria were injected into the plants the growth of the fungus was inhibited and the growth of the cells was arrested even after they attained the size of ½ inch. The destructive action appears related to the presence of the bacteria as the filtrate from the cultures of the various bacteria had no apparent effect.

In 1936, Levine (378) reported that a bacterial parasite had been found throughout the valley of the Mississippi that would destroy uredosposes of *Puccinia* *graminis.* The organism was isolated from garden slugs and appears to be widely distributed in the United States, having been found from Oregon to West Virginia and Minnesota to Mississippi in the wheat-growing areas. It is a species of *Bacillus* and appears to grow best at high temperatures.

Alexopoulos (22) found that the cultures of *Actinomyces albus* inhibited all fungi against which it was used. Among the fungi which it inhibited were *Glomerella cingulata, Physolospora cydoniae, Gleosporium roseum, Colletotrichum lindemulthianum. Bacillus subtilis* was found to inhibit five of the fungi and *Serratia marcesens* to inhibit three. Chester (105) reports that there are substances in the soil that exert an inhibitory action on *Helminthosporium sativum.* It has been shown that *H. sativum* spores will not germinate in soil solution from fallow land but will do so readily in solution of sterilized soil. Chester offers the suggestion that saprophytes can build up the fallow land at the expense of the parasites and that the toxic substances will act against the *Helminthosporium* spores until such time as wheat, or related crops, are grown on the soil and the parasite can build up again. It is generally agreed that root rot is less severe on fallow soil than on soil that has been cropped for some time to wheat or barley.

Some soil organisms are known to be antagonistic to such fungi as *Pythium* and *Rhizoctonia.* Organic matter is an aid to the growth to these saprophytic organisms and the reduction in plant disease may well be responsible for some of the increased yields secured from the use of fertilizers. One of the possible evil effects of the chemical soil sterilizers may be that they kill off the saporphytic organisms which are producers of some of the antibiotic substances but do not increase the forms that control the pathogens. Plant disease bacteria are facultative parasites in the soil but few survive long without the host plant. Evidently they cannot compete with the soil saprophytes. It may be that the bacteriophage may play a deciding role in this portion of the drama of life. But there has been one practical example of the control of a dangerous plant disease by the use of manure. In Texas of the U. S. A. (336) cotton root rot has been successfully controlled by the use of manure. There spoiled alfalfa hay has been used over a period of some years and the root rot has been almost completely eliminated from the area to which the hay has been applied. The interpretation is that in this case the increased organic matter favoured the soil saprophytes against the plant disease organism (*Phymatotrichum omnivorium*).

The host plant itself may secrete an inhibitory substance which is toxic to the pathogen. Berridge (56) found that the sap of potatoes contained a substance which possessed some agglutinating power over the cells of *Bacterium tumefaciens, B. solanasaprus* and *B. phytophthorus.* Thomas (802) found that the galls of the olive, formed as a result of the attack of *Bacterium oleae* contain a substance which will inhibit the growth of the bacteria. He also reported that an agent could be isolated from the centre of spots on leaves of maize caused by *Bacterium stewarti* which was lytic to the bacteria. When *B. stewarti* was grown on extracts of wheat, rye, oats, Kentucky blue grass, fox-tail, apple, celery, crimson clover, tomato, cabbage, peach, boxwood and carrot, the lyric principle could be isolated from each. This may offer clue to the defensive mechanism of the higher plants.

Bacteriophage

In 1915 the first report of the lytic principle which could destroy bacteria was made by F. W. Twort while investigatting the ultramicroscopic viruses. In 1917 d'Herelle made a similar discovery while studying the stools of patients recovering from dysentery. According to Muncie and Patel (507) the first lytic agent was really isolated in 1924 from a diseased cabbage by Mallman and Hemstreet. These authors reported one from *B. carotovorus* at the same time. They found that dilutions as great at 10-17 would inhibit the growth of the organism in broth cultures. A little earlier than this a bacteriophage specific for the legume nodule organism, *B. radicicola* Beij., had been isolated in Holland and the first specificity for plants was demonstrated.

In 1928 a bacteriophage was isolated from the soil at the foot of a peach tree previously infected with the leaf spot caused by *Bacterium pruni* EFS. In 1925 a bateriophage was isolated from a carrot rotted with *B. carotovourus* which proved specific for *B. atrosepticus* and *Ps. tumefaciens* as well. After nine months, however, the agent could cause clearing of only *B. carotovorus.*

A bacteriophage specific for *Ps. temefaciens,* the crown gall organism, was isolated by Israilsky (300) from a crown gall on sugarbeet. But two of the 11 isolants from the same gall proved to be resistant to the lytic principle. Later he was able to isolate resistant strains of the organism from the broth in which the lytic principle was incubated. At that time, however, he was unable to isolate the lytic principle from pure cultures of *Ps. temefaciens.*

In 1929, Brown and Quirk (86) found that the activity of the bacteriophage appeared to be correlated with the pH of the medium. If the organism was active the pH was changed from alkaline to acid.

In 1930, Muncie and Patel (507) reported on their work with the bacteriophage isolated from *Ps. tumefaciens* which was isolated from galls on raspberry and on sugarbeet. They found that from all of the sources the lytic principle caused complete lysis of the bacteria when grown in broth cultures.

Literature is full of instances of the isolation of specific "phages" and from a wide range of host plants and pathogens. Two of the organisms found in India for which specific "phages" have been found are *Bacterium solanacearum* and *B. citri.* These have not been worked on much in India but as they are common it is reasonable to suppose that the lytic principles are also to be found. Franssen also reports the finding of a bacteriophage specific for the black arm disease of cotton caused by *Ps. malvacearum* in cotton seed in Java.

It would appear that the bacteriophage is as widely distributed in nature as the bacteria themselves and no doubt some of the difficulty encountered by early workers in isolating bacterial plant pathogens, was due to the interference of some specific "phage" for the organism in question. In fluid media it manifests itself by causing a clearing of the cloudy or milky appearance of the culture. This clearing is due to the destruction of the bacteria for which it is specific. It has been shown to be active at very great dilutions. Muncie and Patel (507) found that the "phage" they worked with was still active at dilutions as great as 10-21.

The bacteriophage is often referred to as a filterable, or a non-filterable, principle, as the case may be. It can be filtered through candles of porcelain or diatomaceous earth but its passage through semipermeable membranes depends upon the charges of the "phage" particles and that of the membrane. In some cases the basic filters would retain neutral solutions of the "phage" whereas acidified "phage" was retained by the ordinary Berkfeld filters. Both living and dead susceptible bacteria will absorb the "phage". There appear to be antigenic properties possessed by the lytic agents as it is capable of producing specific antibodies when injected into the bodies of animals, and sera obtained from such injections have been shown to be capable of neutralizing the activity of the "phage".

Resistance or non-resistance to the activity of the "phage" is also an interesting phenomenon. It may take place in the natural condition and also in culture. Just what this is it does not appear to be well understood at this time. It may be a case of selection or it may be mutation. But it has often been found that after a culture has been apparently clear growth would start again and the resulting strain would be found unaffected by the lytic agent. It may be that the culture was not pure in the first place, either for bacteria or for the "phage", and the result was a selecting out of the resistant forms. In some cases the surviving bacteria are even more virulent than the first strain and where this occurs there can be no argument for the value of the "phage" as a controlling agent usable against the pathogen. On the other hand, there are also cases where the virulence of the bacteria has been reduced or completely destroyed.

Modern Trends in the Development of Fungicides

Over the world today there are being developed a large number of new compounds to replace the older chemicals that have been standard for so long. Many of the old standard chemicals have never given complete protection against fungus diseases but they were all that were available and thus seemed to be the only hope of the farmer. The older standard chemicals were almost all entirely inorganic in nature. Copper, sulphur, mercury, zinc, being the most common. The new fungicides are likely to be organic in nature and much more specific in their range of usefulness. That the older fungicides failed in the past under some circumstances was due to the fact that they were expected to do too much and perhaps the striking thing to be observed is that they do so well.

Horsfall (in Agricultural Insecticides and Fungicides News. Vol. 4 p. 4-5, 1946) recently remarked that the new developments in fungicides means that the farmer must brush up on his chemistry to be able to understand what is going on. Dr. Horsfall goes on to suggest that the new fungicides offer three possibilities; (1) theraphy, which is to cure; (2) artificial immunization, which means help for the plant to ward off the disease and (3) protection, which means to prevent the plant pathogens from successfully attacking. To cure is difficult. To immunize is still in the future. Protection has received most of the efforts of the plant pathologist.

To-day laboratories the world over, are producing and testing thousands of compounds seeking one that will combine effective control with safety for man and plant. Laboratories have adopted the slogan of Edison and others of "Try and then try again". At one laboratory in the U. S. A. over 6,000 different compounds have been tried and tested. Out of all of these only a few gave any promise. Multiply that by many times, for there are many laboratories working in the U. S. A., and one can get some idea of the amount of work being spent in the production of the modern fungicides. Of course not all of the work is for fungicides alone but they are a product, or a by-product, of the industry.

Most of the new compounds being produced are for the spraying on the plants with the aim of killing the spores directly. But before a fungicide can be released it must be tested so that there is no danger of injury to the host plant. This means that there is much work to be done before release. The chemist, like the architect, must know the structural materials which he is using. He must know the structure of the various fungicidal compounds so that if he finds one similar to them he may surmise that it might also be a fungicide. Then begins the work of testing. If the compound survives the thorough laboratory screening it is taken to the greenhouse and used on various plants to see what the effect will be. If there it proves to be harmless to host plant then it goes to the field for trial under field conditions. There the effect of weather, soil and biological factors are studied in relation to the fungicide. In the field a large number of plants are used and a wide range of diseases tested against. After this it goes to selected farmers for trial under farm conditions. If still successful it is ready for commercial production.

The newer fungicides have been tending toward the organic and away from the inorganic. Some of the new compounds are based on ammonia compounds. contain phenyl and some are of the quinod group. Many of them have long names, for, at present, they have been given descriptive names. At this time they seem impossible for the beginner but before long they will bear familiar short names. For example, tetra chloro-para-benzoquinone is known by the trade name of "Spergon". Mercury phenyl cyanamid is known on the market as "Barbac C". One of the naphtyl acetic acid compounds is known as "Methoxone". The latter is, however, a weed killer rather than a fungicide.

No list of fungicides at this time would be reliable for long. Almost before the print would be dry some of the list would be displaced by new compounds better and more effective for specific diseases. Nor is this the only field in which the chemist is active. From results already obtained, it appears likely that, in the near future, fungicides may be available that will prevent the growth and sporulation of the parasite and thus be a step ahead of any thing available to-day.

Quarantine and the Prevention of Plant Disease

Introduction

From the time man first began to carry plants from one part of the world to another he has carried plant diseases along with them. In this way diseases have become universal. The introduction of a disease from one country to another has often been far more serious than the diseases which have been native. The absence of a disease from any one section means that there has been no selection under natural conditions for resistance and the probabilities are that upon the first introduction losses to crops will be very heavy. The downy mildew of the grape, introduced into France from America, played havoc with the vine industry of that country until hybridization with resistant American stock offered a solution. The American Chestnut was nearly completely destroyed by the blight fungus (*Endothia parasitica*) which was introduced into that country from Asia.

The number of plant pathogens capable of attacking any single crop plant is large. McCubbin (428) has summarized the literature in the field of pathogenic fungi on the cultivated crops and offers some interesting data. The number of known pathogens on rice is 111. Those on sugarcane number 417 and those on the rubber plant (*Hevea*) number 291. These are illustrations of the number which can be listed on the common crop plants. Data from the United States Bureau of Plant Quarantine for the year 1940 showed a record of 25,839 interceptions that could be referred to 325 species of fungi, bacteria and nematodes. Among the examples given was that of potato scab (*Actinomyces scabies*) which was intercepted 469 times from 37 foreign countries. *Puccinia graminis* was intercepted 157 times on products from 18 foreign countries. Apple scab (*Venturia inequalis*) was intercepted 189 times from 37 foreign countries, and Citrus canker, (*Bacterium citri*), was intercepted 17 times on four foreign countries products. The vigilance of the quarantine officials means a great deal to the farmer of the country for experience has taught lessons that will not be forgotten soon. Failure to prevent the introduction of plant diseases and pests has been responsible for the destruction of whole industries which have been built up around certain crop plants.

Water barriers offer some safety from the introduction of foreign plant pathogens but air-borne spores of plant pathogens have been found far out over the bodies of water. Spores were picked up over the Mediterranean Sea as much as 700 miles from land. Lindberg found a few spores of fungi in the region of the North Pole. The air over the centre of the oceans has been found relatively free from the spores of plant pathogens. Mountain barriers also are somewhat effective in preventing the spread of plant disease. Prevailing winds may be effective in preventing the introduction of plant pathogenic spores as well as being responsible for the introduction of spores into a country. Cold loving plant pathogens are not found in the warm regions of the south and vice versa. Humidity and temperature have been found to limit the range of plant pathogens.

The program of disease exclusion requires some four kinds of defence. 1. A knowledge of the pathogen; its identity; likely source and plant materials on which most likely to be found. 2. Strict and thorough control of all plant imports that are likely to be carriers. 3. Adequate domestic survey facilities that will at once detect any pathogen that escapes the quarantine. 4. Adequate organization to either eradicate, or, where that is impossible, to quickly limit the pathogen to a small area.

This involves a knowledge of foreign disease which can be secured only by study and, preferably study in the countries from which the pathogens are likely to come. The acquiring of such knowledge can be greatly facilitated by a free exchange of scientific literature and the exchange visits of those associated with the plant disease surveys and quarantine offices. For the control over the entrance of plant material containing disease, the law must be such that an embargo can be quickly enforced against any or all parts of a shipment of material known to carry plant disease pathogens. It must be capable of being made quickly effective against the shipments of plant materials from any or all countries known to have the plant pathogens.

If the country, from which the shipment has been received, has an inspection service, the material must be accompanied with a certificate of inspection showing the full characters of the material.

The Indian quarantine laws date back to the "Destructive Insects and Pests Act" of 1914. In that act rules were laid down to prevent the import into British India of any insect, fungus or other pests which may be injurious to crops. "Crops" in this case being intended to include all agricultural or horticultural crops and all trees, bushes or plants. By "import" is meant the bringing, or taking by sea, land or air, across any customs frontier defined by the Central Government. By "infection" is meant infection by any insect, fungus or any other pest injurious to crops. By "British India" it shall be construed to mean British India and Berar.

The Act provides that the Central Government has the power to regulate or restrict the import into British India, or any part thereof, or any specified place therein, of any article or class of articles likely to cause infection to any crop or of any insects likely to infest the crops. Such action on the part of the Government must be published in the Gazette of India.

The Central Government may prohibit or regulate the export from a province, or the transport from one province to another in British India, of any article or class of articles likely to cause infection to any crop or of insects generally or any class of insects.

The act further states that after the notification of an embargo on plant materials which may carry disease or pests the railway station or the inland steam vessel station operatives shall refuse to transport such materials.

The Act applies also to the shipments of materials from the province into the Indian States providing that the Indian States prohibit the shipment from the Indian States into the Provinces.

The Central Government may make such rules governing the shipment of materials suspected of carrying diseases and pests, as deemed necessary. Such rules must, however, be published in the official Gazette. The Provincial Governments may make rules effective within the provinces concerned which are in line with the rules laid down by the Central Government. Provincial Governments may fix fines for breaches of the rules up to as much as Rs. 1,000.

The rules regulating the import of plants, etc., into British India, have been revised from time to time; the last such revision being in 1944. Quarantine offices have been established in the ports of Bombay, Calcutta, Cochin Dhanushkodi, Karachi, Madras, Negapatam, Port Blair and Tuticorin. The quarantine inspectors are instructed to see, not only to the shipment of plant materials themselves, but also to all wrappings and packing materials. The latter have been responsible for the introduction of many diseases into one country from another. The Act prohibits the shipping into British India of any plant materials by letter or post, excepting sugarcane which may be shipped to the Government Sugarcane Expert in Coimbatore for experimental purposes. Shipments are prevented by air except in the case of insects or plant diseases which are specifically intended for research and they must be accompanied by special certificates which state the designation and the purpose of the shipment. Where living material is being shipped for research they must be accompanied by a certificate stating that they may be shipped without fumigation, otherwise they will be fumigated before being allowed to go on. In the case of insects, a special certificate, from the Imperial Entomologist, must accompany the shipment if the material is to be used for research purposes. There are a number of special exceptions which are provided in the Act which permit the shipment into British India of certain materials, such as rubber plants and horticultural plants, provided such materials are to be used for research and each shipment is accompanied by a certificate, from the responsible Government officer, stating the designation and the purpose of the shipment.

Potatoes, unmanufactured tobacco and vegetables intended for food may be shipped into British India from Burma without a certificate but all other plants must be accompanied by an official certificate that they are free from injurious insects and disease.

Burma is the only country from which potatoes may be shipped without a certificate. Shipments from all other countries must be accompanied by an official certificate stating clearly where they were grown and that no wart was known in that district for at least twelve months prior to the shipment. Exceptions to the rules are made in the case of potatoes wanted for experiment purposes by the Imperial Economic Botanist of the Imperial Agricultural Research Institute, New Delhi.

Rubber plants may not be imported into British India unless they are accompanied by a certificate showing origin and that they are free from disease. Unmanufactured tobacco may not be imported from any country, except Burma, already referred to, unless accompanied by a certificate showing it to be free from the tobacco moth (*Ephestie elutella*) or that the pest does not exist in the country of the origin of the tobacco. Citrus plants, such as lemon, lime, orange, grape-fruit, or their cuttings shall not be imported into British India unless such shipments are accompanied by a certificate stating that the material is free from the Mal secco disease, caused by *Deuterophome tracheiphila,* or that the disease does not occur in the area from which the shipment has come.

The importation of sugarcane from the Fiji

Islands, New Guinea, Australia or the Philippines into British India is prohibited. Sugarcane shipments from all other countries must be accompanied by a certificate stating that they are free from cane borers, scale insects, white flies, root diseases of any form, pineapple disease (*Ceratostomella paradoxa*), sereh and cane gummosis and that the specimens were obtained from a crop free from mosaic disease and, the Fiji disease of sugarcane does not occur in the country of export. In the case of canes being shipped to the Government sugarcane expert, Coimbatore, the only requirement shall be that they are free from the Fiji disease. Rubber (hevea) plants and seeds shall not be imported into British India from America or the West Indies except by the Director of Agriculture Madras Presidency. Seeds of flax, berseem and cotton shall be imported only by sea and then only if accompanied by a certificate from the Department of Agriculture. The importation of Mexican jumping beans shall be absolutely prohibited. Unginned cotton shall not be imported into British India except from the port of Kathiawar. Cotton seed shall not be imported by sea except for experimental purpuses. In that case it must be accompanied by a certificate that it has been treated so that neither seed nor container carry pests. If there is suspicion that the seed may contain pests or disease the officer at the port of entry may examine and fumigate if required.

There are many other rules which govern the entry of plant materials into British India and the student is urged to secure a copy of the Destructive Insects and Pests Act of 1914 and the rules made thereunder by the Central Government and read them carefully. This is especially important if one is entering the service of the Government of India or of any of the provinces as a plant pathologist or entomologist or will be in any way connected with the shipping or receiving of plant materials likely to carry plant disease or insects. A copy of the Destructive Insects and Pests Act, 1914 may be secured from the Manager, Government of India Press, New Delhi. Under the new Government many changes and additions will occur but it is likely that the main principles of the act will be retained.

CHAPTER V

WEATHER AND THE PLANT ENVIRONMENT

In this chapter the weather and the factors which make up the commonly accepted idea of environment will be discussed.

Weather and Plant Disease

What do we mean by weather? The average man would probably reply that it is the wind, the rain, the heat, the cold, the sunshine and the clouds. Humphreys (297) in his excellent book on the *Ways of the Weather*, which all may read and enjoy, has included a clever verse on the weather:

What is it that moulds the life of man?
The weather.

What makes some black and others tan?
The weather.

What makes the Zulu live in trees,

The Congo native dress in leaves,

While others go in furs and freeze?
The weather.

Our ideas of the weather are often not very clear. Asked to give a definition of weather and we are not able to make a good one. Usually we try to define it all at once. It is not easy to do, for weather is not a single factor. Humphreys (297) remarks that it is often said that the "climate of a given place is the average weather." But as he further states, that definition is useless for it does not really define anything. If we say a wet climate, a dry climate, a warm climate, a climate that is cold, then we begin to mean something.

Ancient man considered that weather was the smile or frown of the gods and their pleasure or anger was reflected in the kind of weather. Man has recognized the influence of weather on his life for many centuries. The farmer has blamed the weather for his ill-luck or good luck. Humphreys (297) has included a little verse that pictures the farmer and his weather better than any we have seen:

What makes some glad and others sad?
The weather.

What makes the farmer hopping mad?
The weather.

What puts a mortgage on your land,
Or makes you sweat to beat the band,
Or takes it off before demand?
The weather.

Although weather and crops have been associated together in the minds of men for ages, it is only within the more recent times that there has been any real effort to determine the actual relationships. Human welfare, disease and weather have been associated together for ages. The ancient Greeks associated the wind and disease. Alexander the Great observed that when he camped near a swamp, a swamp disease appeared to come with the wind. The mosquito and malaria were not associated together at that time. Theophrastus, the great botanist, associated the blast of grain (probably rust) and the wind. But these ideas were based on superstition and backed only by observations. It is just within the past few years that there has been any real attempt to make a careful study of the factors of weather and their effect on plant disease.

It has been recognized for a long time that the spores of fungi are well adapted for wind and water dispersal. Stakman and Christensen (735) have given some exceedingly interesting and informative figures on the number of spores which may be produced by a single fruiting body of such a fungus as *Daedalia confragosa* (Bolt.) Fr., or of *Fomes applanatus* (Per.) Gill. The figures for the former were 682,000,000 and for the latter 5,460,000,000. They report that the apple scab fungus (*Venturia inequalis* Cke.) Aderh. may produce as many as 8,108,200,000 ascospores under a single apple tree in a single season. That a single wheat kernel may contain as many as 12,000,000 spores which would mean 5,000,000,000,000 spores in an acre of wheat where there would be as much as 1 per cent infection.

The ability of a fungus to produce spores depends to a large extent upon the weather. Temperature and humidity are the two most important factors that operate for or against spore production. Closely associated with them is the matter of food availability which may be either dead or living organic matter.

The ability of the fungus to produce further infection will depend upon several factors, such as viability of the spores, susceptibility of the host plants, temperature, moisture, wind, etc., together with the time of the spore discharge. Any one of these factors may be the limiting factor which can prevent the further spread of the fungus.

Dissemination of spores may be local or distant. Examples of plant pathogens that are locally disseminated, would be late blight of potatoes, downy mildew of bajra and maize, most of the smuts and the leaf sports, caused by such fungi as *Cercospora*, *Helminthosporium* and *Phyllosticta*. During the collecting of the data for the late blight forecasting service, Melhus (479) has found that infections would be found up to some 200 yards from the refuse piles of cull potatoes and that these infections would be in the direction of the prevailing winds. This might indicate that the rate of spread might be slow but observations and records of epiphytotics of late blight in various parts of the world indicate that spread may be rapid under some conditions. In the case of downy mildew of bajra, Weston (928) found that the conidia were produced at night and disseminated during the night hours. He determined that spread was mostly local but he was unable to account for the fact that there would be wide skips between infected fields. In some cases the fields would be on different islands. The spread from one area to another, involving great distances, would involve risks that the spores would be killed by the direct sunlight of that season unless the spread took place at night. Such spread must also be dependent upon the growing of susceptible crops in the line of the prevailing winds. For crop disease like late blight, the downy mildews, leaf spotting fungi of the *Cercospora* type and other similar fungi, the normal spread may be expected to be from plant to plant within the same area, from area to area and from field to field. The classical illustration of the long-range dissemination of fungus spores has been found among the rusts. In India the work of Mehta (451-456) and his co-workers has produced some exceedingly interesting information. His conclusions, that the uredospores of wheat rust do not survive on the plains and therefore must come from the hills for each season's infection would indicate, that some, at least, must travel in the air for over 500 miles. Meier (460) determined that the spores caught in the air over the Mediterranean Sea would have to travel over 500 miles to the place where they were caught. In this connection the student should read the article on aerobiology by Stakman and Christensen (735). The authors have given an excellent account of the spread of stem rust of wheat in the United States. In that particular case the spread of the epiphytotic was due to a number of factors. The prevailing direction of the wind determined the general direction of the epiphytotics from season to season but the most serious was the result of a reversal of the wind direction, for a period, which resulted in the accumulation of an unusually large amount of inoculum so that when the normal wind direction was resumed the enormous number of spores produced resulted in the production of one of the worst rust seasons recorded for the plains area of the United States.

The collection of weather data with reference to plant disease forecasting and plant disease control is of relatively recent origin. The past few years have seen special emphasis placed upon the effect of wind, rainfall and humidity upon the dissemination of plant disease. But other factors are also of importance. Melhus (479) in his forecasting of late blight emphasized the importance of temperature as a factor in the spread of the late blight pathogen. *Phytophthora infestans,* the late blight organism, grows best at a temperature of 70° or below. When the temperature rose about 70°F. the host plant had the advantage because the fungus is a cool temperature organism. From the weather data collected in eleven states of the Upper Mississippi Valley and two of the Canadian Provinces, it was possible to determine conditions necessary for an epiphytotic and forecast when such conditions would most likely occur.

Chester and Larsh (107) made forecasts on the probable rust epiphytotics of *Puccinia triticina* in the southwestern plains sections of the United States. To be of practical value, data of this kind must be collected promptly, sorted and evaluated and then distributed to the areas likely to be most interested. Melhus (479) often received the late blight data from the various co-operators by wire. This made it possible to collect the data, organize them make graphs of the temperature and humidity curves for the various areas and then send these data, together with any forecasting that might be possible, out to the affected districts in time for any needed protective measure to be taken before the epiphytotics actually developed. In this way the weather information was in the hands of the growers within a few days or hours, of the time it was sent in to the central station, Methods of this sort have been in use by the various fruit-growing associations of the United States for a number of years. Entomologists have also made use of such weather information in planning moves to prevent damage from insects such as locusts, chinch bugs and army worms. Where large areas of land are planted to the same crops, which are attacked by the same insects and fungi, this type of service offers ever increasing good. Local areas are not in a position to know when and where to expect attacks of insects and fungi unless they have access to some such information.

Weather is largely local and is rarely the same over a very large area at any one time. Distances of only a few miles may make a difference in the extent of damage caused by pathogens. On the other hand, most of the insects and plant pathogens, like the army worm or the rusts, are influenced in their spread by the major weather factors and thus the need of the data collected by the weather bureaus. Newspapers and radios are the most effective means of dissemination of the information.

At Allahabad, rainfall, humidity and temperature data have been collected for a number of years and recently these data are being used in an attempt to explain the behaviour of some of the fungi found on the farm crops. The average humidity and temperature and the total rainfall for the months of June to March, inclusive, are shown in the following tables. During the kharif season of 1945, species of *Choanephora* were found growing on a variety of crops. In some cases they were definitely behaving as pathogens. Although usually found only on the blossoms of brinjal and pumpkins, in 1945 they were observed growing on

Weather data collected at Allahabad, India during the years 1940-1948

Humidity

Year	Month									
	June	July	August	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	March
1940-41	48.30	78.00	83.70	73.50	58.00	52.20	71.80	78.96	66.58	41.55
1941-42	63.56	70.36	83.62	83.20	80.60	53.42	89.96	80.22	77.65	59.76
1942-43	49.08	86.24	79.25	79.52	76.08	74.00	81.03	61.77	75.70	51.59
1943-44	56.80	72.60	93.24	88.00	86.95	73.89	78.84	81.62	81.66	78.80
1944-45	54.70	89.60	88.55	78.76	78.86	69.83	53.80	58.60	..	..
1945-46	53.00	60.65	67.47	76.12	75.50	67.44	60.70	58.30	64.89	52.68
1946-47	56.23	83.50	90.80	86.86	80.30	77.86	81.56	81.02	78.25	56.44
1947-48	55.4	87.3	89.76	87.58	71.06	69.66	..	..	..	..
Average:	54.52	77.28	83.66	82.13	76.61	66.93	73.95	71.49	74.12	66.80

Weather data collected at Allahabad, India during the years 1940-1948

Rainfall

Year	Month										Total Rain-fall
	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	March	
1940-41	0.80	4.37	14.72	5.60	0.12	0.02	0.00	1.03	0.20	0.00	26.86
1941-42	2.74	5.72	5.28	8.65	0.04	0.00	0.00	3.96	4.30	0.36	31.05
1942-43	4.52	7.10	9.98	8.22	0.00	0.00	0.16	0.57	0.22	0.00	30.77
1943-44	0.80	8.29	19.78	8.78	0.74	0.00	0.00	0.00	4.58	4.38	47.45
1944-45	1.09	8.64	19.04	4.98	2.09	0.00	0.90	2.00	0.00	..	38.74
1945-46	1.21	5.62	11.65	2.91	2.97	0.00	0.00	0.00	1.00	0.00	25.36
1946-47	3.69	10.60	12.61	1.29	0.75	1.13	0.08	0.29	0.34	0.45	31.02
1947-48	3.04	14.57	10.14	6.82	0.78	..	..	..	..	..	..
Average :	2.12	7.19	13.01	5.77	0.96	0.16	0.16	1.12	1.52	0.86	32.89

Weather data collected at Allahabad, India during the years 1940-1948

Temperature

Year	Month									
	June	July	August	Sept.	October	Nov.	Dec.	January	Feb.	March
1940-41	93.72	83.38	83.19	83.45	80.30	69.48	63.00	60.78	64.64	75.95
1941-42	89.60	86.40	83.70	83.20	80.10	67.63	62.22	59.76	64.80	75.60
1942-43	94.34	83.75	82.28	81.72	78.50	68.23	60.85	66.07	63.14	75.92
1943-44	92.88	85.63	80.80	83.50	76.84	67.89	61.28	60.54	62.54	73.70
1944-45	82.65	86.95	81.33	79.45	74.62	68.38	62.60	59.56	68.19	..
1945-46	84.30	85.90	86.32	82.75	75.92	65.66	59.70	61.91	64.34	72.45
1946-47	87.93	86.83	83.30	83.00	75.90	68.00	63.24	58.01	63.09	76.19
1947-48	91.2	84.4	83.4	83.18	76.82	68.64	..	..	..	..
Average	89.33	85.55	82.99	82.44	77.45	67.89	61.85	60.94	64.55	74.80

wheat seedlings in the field, on sum hemp flowers, on castor leaves, on guava leaves, on bajra spikes and on species of *Amaranthus* in the variety garden. In 1946 *Choanephora* was found only rarely and then only on the petals of brinjal and pumpkins. Seeking an answer to the difference in the amount of the fungus growth in the two seasons, one would naturally turn to the weather. There is a slight difference in temperature but hardly enough to cause the difference in the appearance of the fungus. There is a difference in the distribution of the rainfall and in the total amount and there is a striking difference in the average humidity. The average difference in humidity for the months of June to November, inclusive, for the years 1945 and 1946 was 12.56 per cent. That is, the humidity during the season when *Choanephora* was more or less pathogenic, averaged 12.56 per cent lower than the corresponding months of the season when it did not occur.

During 1945-46 no rain fell during the months of November, December and January. These are the critical months for rusts. The first rusts to be observed in the vicinity of the Institute farm were found on a village farm near the Institute on February 9. At the same time all three of the common wheat rusts were found on the Institute farm. Rust may be expected to appear in normal years any time after the first of December. This was some two months after the normal appearing time. It is hardly likely that no rust spores fell into the fields during this period. But with no rainfall and a low humidity the conditions were unfavourable for germination of the spores and the infection of the grain.

Using the data collected during the kharif season of 1946, it was possible to predict the appearance of the rust for that season almost to the day. Actually the first rust was observed on December 9 and predicted for the end of the second week of that month. One thing which was not predicted was the absence of stripe rust for the 1946-47 season. Stripe rust was fairly common during the 1945-46 season but was not observed during the 1946-47 season. Further observations will be needed before this fact can be reliably explained. The same must be said for the pea rust (*Uromyces fabae*) and for alfalfa rust (*Uromyces striatus.*) which were unusually severe in 1946-1947. Powdery mildew (*Erysiphe polygoni*) was also very serious on the field peas and *Erysiphe cichoracearum*, the cucurbit powdery mildew, was serious on pumpkins.

Going back over the weather records it seems certain that the most important single factor influencing the activity of these fungi was the humidity, which in turn was the result of a more uniform distribution of rain throughout the season. Carefully collected data over a few years should offer a basis for the explanation of the appearance, or seeming disappearance, of the fungi common on the farm crops. Data already being collected at such centres as the Rust Research Laboratory, Flowerdale, Simla; the Indian Agricultural Research Institute, New Delhi; the Government Agricultural College, Cawnpore; and the Allahabad Agricultural Institute, Allahabad, should be brought together at one central point, studied, organized and then redistributed to these centres for their information and guidance.

ENVIRONMENTAL FACTORS AND PLANT DISEASE

The preceding portion of this chapter has dealt largely with three factors, namely, temperature, rainfall and humidity as associated with weather and plant disease. When these factors are considered in relation to the soil they are usually thought of as environmental factors. This is partly a matter of word usage and partly a matter of definition. It is evident that one must be a continuation of the other and that one cannot change without a corresponding change in the other, although it is agreed that weather will exert the greater influence. Soil factors are complicated and difficult to understand. The soil itself is complex. It is a constantly changing medium. The air is constantly exerting an influence upon the soil. Until the factors constituting the soil environment were analyzed one by one, little progress was made toward understanding them.

One of the first to make a study of the different soil factors was L. R. Jones (322) and his associate, who constructed control chambers by means of which they were able to study the influence of the soil factors upon the root-rotting fungi found associated with the diseases of cabbage, tobacco and potatoes. Dr. Jones observed then, however, that when the factors were studied under green-house conditions one should never expect to be able to duplicate them in the field.

Recently some excellent reviews of certain phases of the root rot disease problems have been made. Students should read the reviews by Simmonds (695), Berkeley (55) and by Cooley (129) as well as the chapter 3 to 8 in Garrett (242). Among the factors influencing the root rot disease fungi, which are of greatest influence, are the soil temperature, soil moisture, soil reaction and soil organic matter. There are other factors such as aeration, cultivation, rotation, etc., which play a part but just how much influence they exert upon the root-roting pathogens is not certain at this time.

Temperature. Changes of only a few degrees will directly influence the activities of many of the soil fungi. The spores of the members of the genus *Fusarium* usually have a rather narrow limit of temperature. Comparisons of findings in the United States and Europe verify these statements. In the temperate regions of North America there are two diseases of tomato, *Fusarium bulbigenum* var. *lycopersici* and *Verticillium albo atrum* which will serve as illustrations of the point in question. *The Verticillium* grows best at 21-23°C. but is inhibited at 25°C. *Fusarium* has an optimum temperature of 28°C. Differences between the temperatures of the tomato-growing regions of England and the United States are sufficient so that in certain areas in England *Verticillium* wilt was much more common than *Fusarium*, whereas it was the reverse in the tomato-growing regions of the United States. The presence of the fungi in the different countries is directly related to the temperatures of the tomato-growing area.

Rhizoctonia solani is another fungus which is a comparatively low temperature organism. Although it is present in the soils of the Allahabad area throughout the entire season, it becomes most active after the beginning of the cool weather has set in, usually after October. From that time on it can be isolated readily from the roots of a large number of the crop plants which include wheat, papaya, potatoes, tomatoes, cabbage and other garden and field crops. *Pythium* species are also cool weather fungi and make their most destructive attacks after the beginning of cool weather. *Pythium* species have been isolated from wheat seedlings, from tomatoes, papayas and a number of other crops grown in the rabi season.

Keshwala (331) determined that *Sclerotium sclerotiorum* will not grow above 30 C. so that it would be considered a cool temperature fungus. Kulkarni (355) studied the cotton root rot organism, *Fusarium vasinfectum*, in Bombay and found that it was most active from 20°C. to 27°C. and that it ceased to grow at 32°C. Chaudhuri and Singh found that withertip of *Citrus*, (*Colletotrichum gloesporioides*) has a temperature range of 15°C. to 35°C. Uppal and Kamat (851) found that *Phytophthora parasitica* will grow at 35°C. whereas *P. infestans* is a comparatively low temperature fungus and will not grow well above 21-22°C. Melhus (479) in his late blight forecasting used 70°F. as the critical point to be watched for while collecting data for the service. Critical temperatures of many of the fungi are known and these may be studied at the same time as the weather data and together they may become the basis for possible predictions of epiphytotics before they actually appear and thus make it possible to take advantage of the time allowed to prepare counter-measures.

Soil moisture. Soil moisture is equally important as a factor in the reaction of the root-rotting fungi. The moisture content of the soil may directly or indirectly affect the host or parasite and thus prove a vital factor in the parasitism displayed by the pathogen. Garrett (242) has made a list of the common soil-borne fungi of the United States and England favoured by high moisture content. Among these are *Sclerospora graminicola, Phytophthora parasitica, Helminthosporium sativum* and a number of species of *Fusarium*, all common to India. Several species of the genus *Pythium* are also favoured by high moisture content but the list did not include any familiar to India. However, *Pythium aphanidermatum* has been shown to be more severe on poorly drained soils and *Rhizoctonia* has been shown to be most virulent in soil with a moisture content of 40% or over.

Among the fungi favoured by low soil moisture content, Garrett (242) listed *Sorosporium reilianum, Sphacelotheca sorghi, S. cruenta, Tilletia tritici, T. levis, Ustilago avenae, U. levis,* and *U. hordei.* These are all common in India. They will be recognized as rabi crop diseases that exist when the rainfall is lowest.

The time of rainfall is also of importance in the incidence of disease. Garrett (242) presents a summary of a 34-year historical survey of the rainfall and prevalence of take-all disease of wheat in South Australia. There, during the months of August, September and October, the amount of the disease was correlated with the amount of rainfall. In Saskatchewan, Simmonds (695), reported that over a 5-year period it was found that the incidence of the same disease of wheat was correlated with the aggregate rainfall during

July and the first half of August. It will be observed that a majority of the fungi that are favoured by low moisture content are smuts. It is believed that the low moisture content hinders the growth of the host plant more than the fungus and thus permits an invasion by the pathogen. This has been shown to be true in the case of seedling blight of maize (*Gibberella saubinetii*) and *Helminthosporium sativum*, the root rot of oat seedlings.

Soil reaction. The influence of the soil reaction on the growth of fungi is well known. Some fungi are capable of growing very well in a high acid soil, while others are very definitely limited in growth by a pH of less than 6-7. Corn root rots and the root rot of sugarcane seem to be able to grow better in the presence of aluminum and are thus more severe in soils containing high percentages of aluminum. Barkeley (55) stated that the absorption of aluminum is correlated with high acidity. Aluminum salts were not found in soils with a pH higher than 5.8 and the root rots were not present in soils of a pH of 6.00. The same author reports work done that may indicate potash has the property of greatly decreasing or eliminating the model accumulation of iron or aluminum in the plant. This has the same affect as the raising of the soil reaction to a pH of 5.8, at which point the aluminum salts become insoluble in the soil solutions.

Ling employed a three-salt nutrient solution, comprising potassium di-hydrogen phosphate, calcium nitrate and magnesium sulphate in cultures used for determining amount of stripe rust (*Urocystis occulta*) on rye and found that the disease was lowest when potassium was highest. Infection was highest in the case of those receiving equal proportions of calcium nitrate and magnesium sulphate but low potassium. Garrett (242) makes the observation that where the development takes place outside of the host plant, liming the soil appears to have a direct effect upon the pathogen, but in the case of the fungi living within the host plant the effect is indirect through the physiology of the host.

Soil organic matter. The organic matter of the soil as a factor in the control of the root disease fungi has received a considerable amount of attention during the past two or three decades. One of the most convincing pieces of work along this line was that of King and Loomis (355) and later by King (356) on the control of the cotton root fungus, *Phymatotrichum omnivorum*) in the southwestern portion of the United States. King trenched the soil to a depth of 10″-14″ with manure at the rate of 15 to 20 tons per acre. This was done during the cool season and the area irrigated immediately. The experiment was begun in 1932 and by 1935 he was able to report the death of only 1.6 per cent of the plants in the trenched soil as compared to 56.2 per cent in the untrenched areas. By 1935 the disease was found in only 9 per cent of the trenched areas whereas it was found in 50 per cent of the untrenched. King attributed this difference to the antagonistic action of the saprophytic manure decomposing fungi. At the Allahabad Agricultural Institute trenching with manure was done in 1938. In the autumn of 1946 barley plants growing on the trenched areas were found to be more free from *Helminthosporium* and *Fusarium* species of fungi than those examined growing on the control areas near by. This was some eight years after the trenching was done.

Chaudhury (121) reported the effect of manuring on the control of sclerotial rot of pan (*Piper betle*). He used mustard oil cake, ammonium sulphate, sodium nitrate and ammonium phosphate. He found that when he used mustard oil cake at the rate of 3936 pounds, ammonium sulphate at the rate of 980 pounds, sodium nitrate at the rate of 1280 pounds and ammonium phosphate at the rate of 1248 pounds per acre he was able to reduce the infection from 31.90 per cent to 2.01, 1.87, 1.90 and 2.09 per cent respectively. The control exerted on the pathogen appeared to be in direct proportion to the amount of nitrogen he applied. Of course that would not mean that it would go on indefinitely as it is well known that the mineral salts will become toxic from concentration if too much is applied.

Mitchell et al (481) studying the effect of organic matter upon the cotton root rot organism (*Phymatotrichum omnivorum*) observed that the fungus appeared unable to stand the presence of organic matter when they added as much as 3 per cent barnyard manure or 3 per cent chopped sorghum to the soil. When these amounts were added the fungus either failed to grow or soon stopped and the mycelium showed disintegration. Chopped cotton stems or roots had the same effect. Microbial populations increased rapidly in such soils. If, however, the soil was sterilized before inoculation, the fungus grew readily. There was also pronounced effect upon the survival of the sclerotia. When sclerotia of *P. omnivorum* were buried in such soil only 30.3 per cent survived in the soil receiving the organic matter, 85.6 per cent survived in the soil receiving the superphosphate and 80.4 per cent in the untreated soil. He found that the chopped sorghum was more effective, though not significantly so, than the barnyard manure. Thus the effect of the organic matter appears to be indirect. Many micro-organisms appear antagonistic to the plant pathogens. The addition of nitrogen may stimulate the fungus as well as the host plant but the difference is in the toxic action of the micro-organisms. Leach and Davy (367) found that when nitrogen was applied to sugar beets *Sclerotium* root rot was decreased, the decrease being in direct proportion to the amount of nitrogen applied. When 50 pounds of nitrogen an acre was applied the root rot decrease was 28 per cent. When 100 pounds an acre was applied the decrease was 54 per cent and when

200 pounds an acre was applied the root rot decrease was 65 per cent.

All of this must not be taken to mean that nitrogen is toxic to the fungus. The fungus uses nitrogen just as the host plant does. In fact it has been found that in some cases when the fungus has gained entrance into the host plant it will not grow unless there is nitrogen present. When the host plant is growing on a nitrogen deficient soil, fungus attacks are less severe than on soils rich in nitrogen. This has been recognized in the case of rusts and mildews for a long time.

Many of the plant pathogenic fungi are saprophytic for much of the time. Where abundant organic matter is available, such fungi as *Fusarium*, *Rhizoctonia* and *Rosellinia* species are largely saprophytic in behaviour. Evidence of the saprophytic nature of such fungi as *Rhizoctonia solani*, *Phymatotrichum ommivorum*, *Fusarium culmorum*, *Ophiobolus graminis* and *Rosellinia arcuata* is presented by Garrett (242).

The longevity of the spores of the root-rotting fungi is correlated with the amount of organic matter in the soil. It was found that the addition of organic matter to the soil reduced the time of sclerotial decay or disintegration of the cotton root rot fungus. In some cases the cotton root rot organism was practically eliminated from soils by the use of organic matter.

Crop rotation. Crop rotation for the control of soil-borne organisms has been practised for a good many years. In former years the farmers secured better crops when they rotated them on the farm but they had no very clear ideas of why they did so. They held that the soil became sick from substances the plants had excreted and therefore must be rested from that crop. In the United States the Upper Mississippi Valley was the centre of the flax-growing industry during the latter part of the last century. It was early recognized that flax could not be grown on the same soil for more than two seasons without the crop becoming run out.

The trouble was called "flax sick" soil but there was no idea of the trouble being due to fungi in the soil. It was not until 1901 that Dr. F. L. Bolley, of the state of South Dakota, was able to show conclusively that the cause of the flax-sick soil was a fungus pathogen, *Fusarium lini.* This opened the eyes of the plant pathologists in the United States and focussed their attention on the soil as a possible source of the organisms which produce plant disease. The discovery of Dr. Bolley was followed rapidly by other similar studies and soon a considerable mass of literature was accumulated dealing with the plant diseases associated with the soil.

The enforced rotation of the land, because of the plant diseases which appeared after a crop had been grown on the same land for more than one season, led to the study of the reasons for the benefits derived from the rest. It had been held that some beneficial chemical action took place during the rest which helped the plant outgrow the disease. It was assumed that the chemical actions released the plant nutrients which were held in the soil complex and thus they became available for the plants. More recent theories, however, are that saprophytic fungi play a very important part in this phenomenon. It is now held that during this idle time, organic matter, in the form of weeds, etc., accumulates and thus the organic matter-decaying bacteria and fungi accumulate. These are in turn antagonistic to the plant pathogens in the soil, which, without the host plants to feed upon, are at a disadvantage and thus unable to survive the period. Thus when the host plant is again planted in that area there will be little of the root rotting for a season. The converse of this is also true. If the same crop is planted on the same field year after year the plant pathogens increase in the soil. This will be especially true if there is no special organic fertilizer applied to help maintain the saprophytic flora of the soil. In 1944, in the state of Iowa, U.S.A. near the town of Roland, a field of canning peas was examined. The field had been planted to peas for three successive years and no special fertilizers had been applied. Over 90 per cent of the plants were dead and when the roots were examined in the laboratory it was found that they were filled with the resting spores of a species of *Pythium*. About three miles from this farm another was visited where a field was examined that had been planted on soil which had been in red clover the previous season. This field was netting the farmer about Rs. 300/- per acre. The difference was that one had followed the sound practice of rotating with organic manure and the other had not. It is beginning to be believed that a sound practice of fertilizing with organic manure will make fallowing unnecessary.

It also appears that some crop plants are also antagonistic to plant pathogens. Bose, writing in *Agriculture and Live Stock in India* in 1938, reported that when tobacco followed pigeon pea, the incidence of wilt (*Fusarium vasinfectum*) was significantly less on the succeeding crops of pigeon pea than when followed by either linseed or fallow. What this benefit is really due to is not clearly understood. Soya-beans have also been shown to reduce some of the soil-borne pathogens. This relationship between the crop and parasite is a field that offers a storehouse of interesting findings to the plant pathologist.

Dispersal of root-rotting fungi. Root-rotting fungi may be dispersed in many ways. The more important are on plant parts, such as root, stem, leaf and seed, by insects, birds, animals, water, man and in some manure. The most important means of dispersal are by plant parts, which may carry these fungi in numerous ways. It is to prevent this type of plant dispersal that the Plant Quarantine Acts have been designed. See section under Quarantine and Prevention of Plant Disease. That fungi may become generally scattered over a farm by means of plant parts, cultural practices and irrigation water, has been illustrated by the findings on the Institute farm at Allahabad. Such fungi as *Rhizoctonia solani, Sclerotium rolfsii,* species of *Fusarium, Pythium* and *Helminthosporium* have been isolated from diseased plants from all portions of the farm. This would indicate that we must replace rotation, or add to rotation, application of organic manure in greater amounts than we have been doing if we would improve our yields.

CHAPTER VI

THE MORE COMMON DISEASES OF WHEAT, BARLEY AND OATS IN NORTHERN INDIA

Disease of the Wheat Crop

Wheat is one of the most important crops of India and therefore it is of importance that the common diseases found on it should be familiar to the student who expects to become an agriculturist. Most of the wheat is grown in the northern portion of India and according to Aiyer 95 per cent of the crop is grown north of a line drawn between Bombay and Calcutta.

The most important diseases of wheat may be said to be the rusts and smuts. Other diseases are of importance but they receive less attention than the two groups mentioned.

In addition to the rusts and smuts, *Helminthosporium* leaf spot, powdery mildew, sclerotium disease, *Septoria* disease, seedling blight, take-all and root rot diseases of wheat will be discussed in this section of the chapter.

The Black Stem Rust of Wheat

Host plant. When all of the forms of *Puccinia graminis* are considered together, the host range includes practically all of the species and varieties of *Triticum, Hordeum, Avena* and many others. However, if only *P. graminis tritici* is considered then the range of host plants is confined to the genus *Triticum.*

Geographical distribution. It is world-wide and appears to have been so from the first cultivation of the wheat plant. Wherever the wheat plant has gone the rusts have gone too.

Appearance on the host plant. The appearance of the rust on all of the major host plants is very similar. The attack may be on the leaf, sheath, glume or stem. It is usually more severe on the leaf sheath and stem.

There are two stages of the rust on the wheat plant: the red rust stage and the black rust stage. The red rust stage (named so because of its brick red colour) is the first to appear and is usually more severe on the leaves and leaf sheaths. This is the stage which does the real damage to the crops. Fields may be red with this stage of the rust and it is not uncommon when the infection is severe to see men, animals and machines coated with the brick-red spores as they come from the harvest fields.

The black rust stage follows the red rust stage and may be intermingled with it. It usually does not appear, however, until the grain is in head and may be delayed even until after the harvest. The red and black rust stages may be found in the same sori or in separate sori. In India the common name is the black stem rust and it is often referred to as the rust.

The organism. Puccinia graminis tritici (Pers.) Erikss, and Henn.

The stem rust of wheat possesses all of the spore forms, two of which are borne on the barberry (*Berberis* spp.) and two on the wheat. That makes the rust a heteroecious form. On the barberry are found the pycnia and aecia, and, if the oogonia are considered, there are really three spore forms on the barberry. The pycnia are flask-shaped bodies imbedded just beneath the epidermis and usually on the upper side, though occasionally one may be found on the under surface. The aecia may be on the upper surface but are usually on the lower surface.

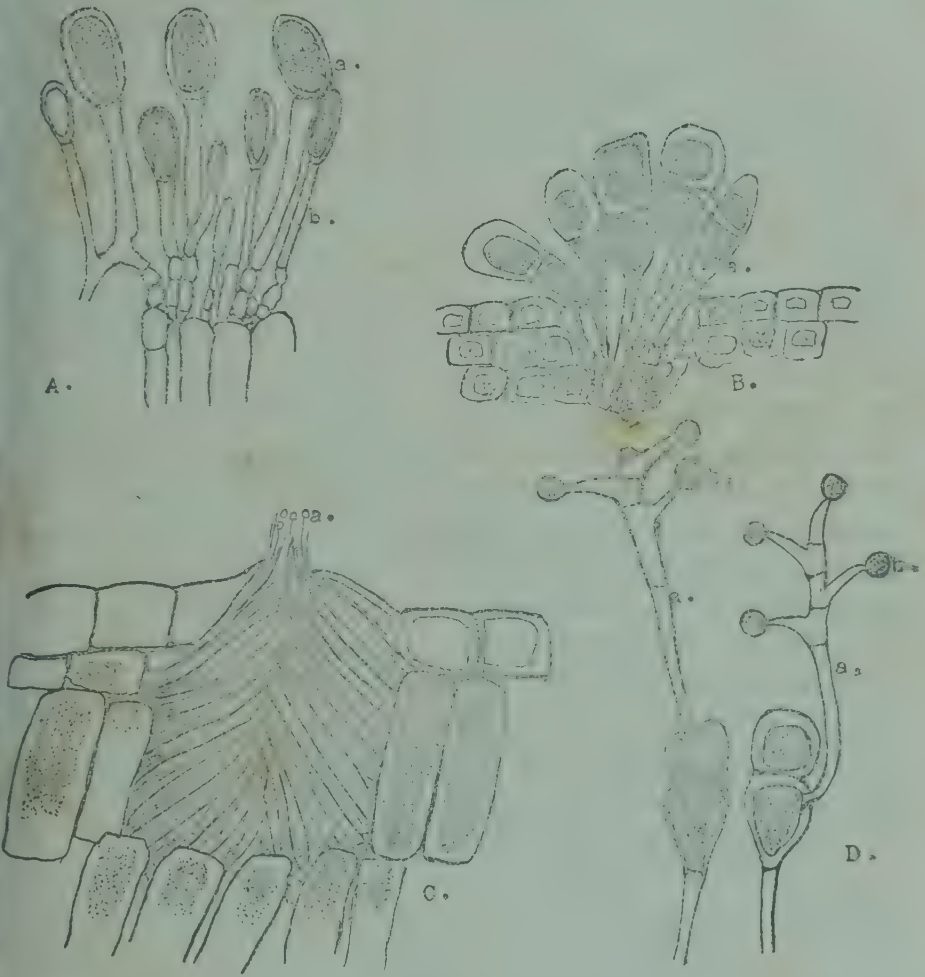
The early conception of the function of the pycnia was that they were true pycnia and the pycnospores were vegetative spores. Later the idea was conceived that they might have had some function in the reproductive process in the past but were now functionless. The work of Cragie (132-137) was the first real evidence that they have a function. This, followed by the work of Hanna (259-263) and of Allen (17-21) proved beyond dispute that they are the male gametes of the rust. Andrus (see under *Uromyces*) was the first one to show the female structure of a rust in action but similar structures have been shown to exist in wheat rust as well. Cragie, working with the sunflower rust (*Puccinia helianthi*), demonstrated that, by sowing a single sporidum (single basidiospore), no aecia were obtained but that if he would sow sporidia of opposite sex on the leaves of sunflower, aecia always developed provided the infections were close together. He found that all pycnospores were uninucleate and produced uninucleate hyphae and he secured binucleate hyphae only when he would sow sporidia of opposite sex potentialities on the same sunflower leaf.

Hanna (260), some two years later, demonstrated that when monosporidial (single basidiospore) infections were produced on the barberry with *Puccinia graminis tritici* no aecia were ever developed but that when mixed sporidial infections occurred aecia always developed. When monosporidial infections were made, they produced uninucleate hyphae but the mixed infections produced binucleate hyphae.

Miss Allen (17) found a structure among the fungus hyphae of a barberry leaf infected with *P. g. tritici* which was very much like trichogyne of a red algae and like the structure described by Andrus (see under *Uromyces*) for the bean rust. She found that when she sowed sporidia on the barberry leaves she secured infections ranging from all spermagonia and no aecia to those developing aecia in all cases. For the most part she found that the infections were about 50-50. This was experimental evidence that the sporidia are in approximately equal number for both sexes. The sporidia are not usually referred to as sexes but as + or — (plus or minus).

After the above evidence had been accumulated regarding the nature of the sex relationships of the rust it was not so difficult to piece together the cycle of events during the period of time the rust lives on the barberry. The sporidia from the germinating teleutospores on old stubble in the field are blown to the young barberry leaves. If spores of opposite sex potentialities chance to fall upon the same leaf and infections follow, the hyphae of the sporidium bearing the male sex potentialities will produce a pycnidium and the hyphae of the sporidium with the female sex potentialities will produce the oogonium which produces the trichogyne-like structure. The tip of the trichogyne seeks the surface of the leaf through the stroma nearest to it and pycnospores are carried to it by insects, water or perhaps by wind. When a pycnospore has become attached to the trichogyne tip the walls between the two break down and the nucleus of the pycnospore is permitted to enter the trichigyne tip and migrate into the base (oogonium) where a union takes place and the fertilization is complete. Binucleate hyphae result from this union and the aecium results soon after. The aeciospores are capable of infecting the wheat plants but not the barberry.

As mentioned above, there are two stages of the rust on wheat: the red rust stage and the black stem rust stage. The red rust stage is the uredospore stage and is sometimes referred to as the summer spore stage. The uredosori are more or less elliptical and brick-red and the epidermis hangs about the edge as a ragged fringe. The uredospores are elliptical or pyriform, the shape differing somewhat with the different forms, although this is not a reliable diagnostic character. They are orange yellow when seen under the microscope, possess four nearly equatorial germ pores, are covered with spines and are borne singly on short pedicels. They are binucleate although both nuclei may not be visible under the microscope at the same time. They measure some 9 by 27 microns.



Diagrams illustrating the life cycle of black stem rust of wheat.

A. Section of a uredosorus. a. Uredospore. b. Uredophore.

B. Section of a teleutosorus. a. teleutospore.

C. Section of a pycnium. a. pycnospore or male gamete.

D. Germinating teleutospores. a. basidia. b. basidiospores.

Teleutosori are similar to the uredosori except for colour. They are dark brown to black and may be longer than the uredosori. The teleutospores are fusiform, typically two-celled with a slight constriction at the septum and each cell possesses a single germ pore. The upper cell pore is at the apex while that of the lower cell is near the septum. The apex of the spore is rounded, slightly pointed or, more rarely, flattened. They measure from 40 to 60 by 15 to 20 microns. They germinate to produce the typical promycelium which become four-celled by division and each cell produces a basidium borne upon a short stalk (sterigma). The production of the basidiospores was discussed under the *Uredinales* and will not be repeated here.

The existence of physiologic forms of the wheat has been known for a number of years. Margaret Newton (543) reported the existence of at least five physiologic forms of *Puccinia graminis tritici* in western Canada as early as 1921. These were identical with forms that were then known in the United States. At the present time there are a number of physiologic races of rusts identified which consist of a still greater number of forms or varieties so that the total number of different varieties, forms, races, etc., is probably in the neighbourhood of 150 (see Dickson (190) pages 163-167 for the complete list of physiologic forms).

Hybridization between varieties, forms, races, etc., has been shown to take place and it is in this way that the new forms appear. Stakman et. al. (730) crossed *Puccinia graminis agrostis* and *P. graminis tritici*, form 36, and found among the resulting hybrids several that were different from both parents. Eight distinct forms were found in the aecidial cups of which three No. 67, 69 and 72 were new to science at that time. Another form, No. 58, was new to the U. S. but was known to exist in Portugal. At the present time there are some seven forms of the black stem rust known in India. These are Nos. 15, 21, 24, 34, 40, 42, 75.

Of these Nos. 15, 21, 40 and 42 are common in the United Provinces.

Life cycle: The complete life cycle of the rust occurs only in the cooler regions where it is possible to find the barberry. Mehta (452) states that it is impossible for the uredospores to survive the hot season on the plains and as there are no barberry plants present it is impossible for the rust to survive the hot season. He believes that the rust must live over on the wild and cultivated grasses in the hills and that each season the uredospores are blown by the wind from the highlands onto the plains and by progressive stages reach all sections where wheat is grown. Since wheat matures in the hills first there is time for the uredospores to reach the fields on the plains and infect them in season. The infections usually appear on the plains sometimes between the 10th of December and the last of January. Sometimes in early December and sometimes the infections do not appear until February, as was the case in 1945-46. The exact time of the migration of spores from hills to plains is not known but Mehta states that the wheat at the foot of the Kumaun Hills shows infection about a week earlier than on the near by plains.

From information now at hand it appears that one will need to trace the life cycle for each major district in India. On the plains it will consist of uredospores and teleutospores with the uredospores being the only important spore form.

Control. At present the only real hope of the farmer is resistant varieties. The existence of physiologic forms makes the production of resistant varieties a difficult task. It means that one must develop a resistant variety of wheat for each section where the physiologic forms of rust differ. At Allahabad, P. 165 has so far shown itself to be resistant to the forms of black stem rust occurring in this area. Mehta states that the vulgare wheats of Kenya are showing resistance to the Indian forms of stem rust and may be the source of commercial varieties for the future.

Recently Hart and Allen (268) found that paratoluenesulphonylamide at the rate of 1 gram per square meter of soil surface and aothotoluenesulphonylamide at 0.8 gram per square meter soil surface gave promise of protecting from stem rust.

Derzhavin crossed *Triticum duram* var. *leucurum* with perennial rye, *Secale montanum*, and produced fertile hybrids with 42 chromosomes. They appeared highly resistant to black stem rust and free from the smuts. They readily crossed with both the hard and soft wheats. It might be of interest in this connection to note that Sax (672) had found that as the chromosome number increased from 7 to the higher numbers of 14 and 21 the resistance to rust decreased but at the same time the economic value increased. Peterson and Love crossed *Triticum vulgare* with *T. durum* var. *imuello* and produced 50 lines of hybrids highly resistant to *P. graminis stritici.* The chromosome number varied among the hybrids but was mostly 42, with some 38 ,39, 40, 41, 42 and 43 chromosome wheats being found.

Leaf Rust or Brown Rust of Wheat

Host plants. Species of *Triticum* and related grasses.

Geographic distribution. It appears to be worldwide. It is the most common rust in India and is the first to appear on the wheat each season although the time of appearance in relation to season may vary. Occasionally it may be later than the stripe rust.

Appearance on the host plant. The first appearance of the leaf rust is usually in December although it may be observed as early as November or, occasionally, not before January. It usually appears two to three weeks before the black and stripe rusts. When first observed the sori are minute round spots, that are nearly orange in colour, and it is sometimes called the orange rust. But as they become older the sori turn brown and from this stage the term brown rust has come into use. In the U. S. the term leaf rust is more common.

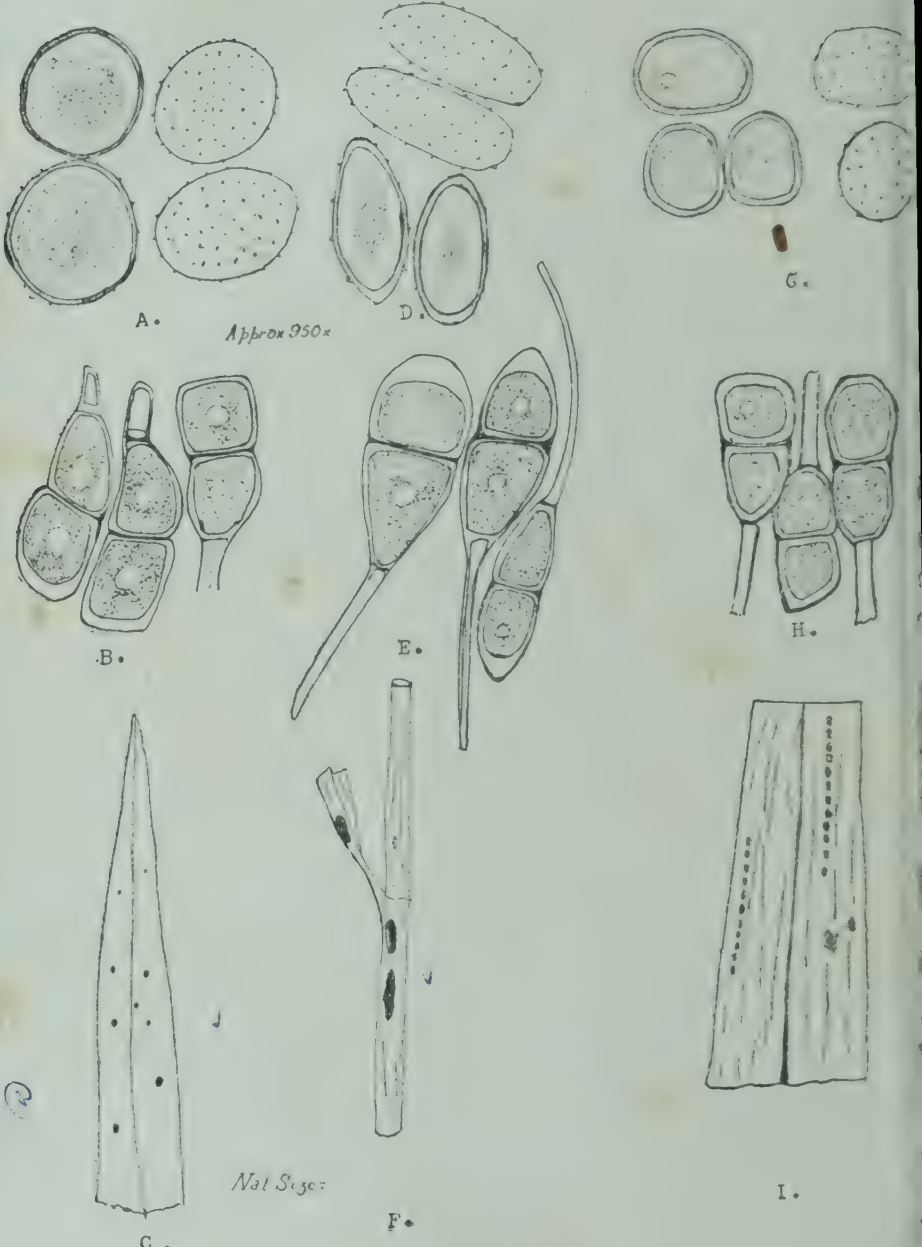
The sori are irregularly distributed over the leaf and this character is an aid quickly differentiating between this rust and the stripe rust. The sori are rarely found in large numbers in the leaf sheaths or stems. If the telia are formed they may be on the leaf sheath. They are darker that the uredia and thus easily distinguished.

The organism. The organism is *Puccinia triticina* Erikss. The brown rust of wheat is heteroecious, the pycnia and aecia being found on species of *Thalictrum.* In India the alternate host is *Thalictrum avanicum* but the aecial stage occurs on this plant in July and August whereas the uredial stage may not occur until January, which would indicate a rather loose relationship between the alternate host and cereal hosts so far as the rust is concerned. This would support the contention that the uredospores carry the rust over the resting period.

The uredosori are round and yellow, or light orange, in colour. becoming more or less brown as they mature. They are never arranged in rows but are scattered over the leaf surface. Although they are found in large numbers they do not run together. The uredospores are nearly round and about the same size and shape as those of the stripe rust but have a brownish wall.

The teleutosori are rare but may occasionally be found on the leaf sheaths. They are more or less broken into compartments by the paraphyses. The teleutospores are similar to those of the stripe rust but more oblong or cuniform in shape, slightly constricted at the septum and with no apical thickening of the wall. Germination is similar to that of the black stem rust.

Life cycle. Where the alternate host plays no part in the life cycle the uredospores carry over on the susceptible host plants in the neighbourhood. Under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D.
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Nat Size

Diagrams illustrating the comparative sizes and shapes of the three common rusts found on wheat.

A. Uredospores of *Puccinia triticina.*

D. Uredospores of *Puccinia graminis.*

G. Uredospores of *Puccinia glumarum.*

B. Teleutospores of *Puccinia triticina.*

E. Teleutospores of *Puccinia graminis.*

H. Teleutospores of *Puccinia glumarum.*

C. Sketch of portion of wheat leaf showing uredosori of *Puccinia triticina.*

F. Sketch of stem of wheat showing uredosori of *Puccinia graminis.*

I. Sketch of portion of leaf of barley showing uredosori of *Puccinia glumarum.*

favourable conditions the mycelium remains viable in the leaf tissue and when this occurs new uredia may develop about the margins of the old ones. When much of the mycelium remains viable there is liable to be an epidemic of the leaf rust. In India it is not thought that the mycelium can remain viable over the hot period so that the spread of the rust each season is thought to follow the same course as that of the black stem rust, i.e., to follow the crops from the hills to the plains.

Control. Resistant varieties appear the best hope of the farmer. It appears that resistance may be due to several factors some of which may be morphological and others physiological. In some cases it has been observed that the rust hyphae may penetrate the stormatal opening but rarely get beyond the infection vesicle stage. In other cases the tissues are killed so quickly that the fungus starves. Waters (913) found that the formation of uredospores took place when the photosynthesis was rapid and that when it was inhibited the production of teleutospores took place. This would indicate that the production of teleuspores is an indication of starvation of the fungus.

Dickson (190) page 180-182, gives a key of the 58 physiologic races of brown rust known at that time in the U. S. A. In India at this time there are some six races known. These are Nos. 10, 20, 63, 106, 107, 108 and 26. The physiologic forms known to be on wheat in the United Provinces are Nos. 10, 20, 63 and 107. There are no immune commercial varieties at this time but I. P. 165 has shown high resistance in trials at Allahabad.

The Yellow or Stripe Rust

Host plants. The stripe rust is found on species of *Triticum*, *Hordeum*, *Secale* and other members of the grass family. The form in India does not appear to attack oats.

Geographic distribution. It is found in both the old and the new world wherever barley and wheat are found. It is common in the Ganges Valley.

Appearance on the host plant. The rust receives its name from the yellow uredosori which are formed in linear rows on the leaves. It is this character which makes indentification easy and the ready differentiation between the stem rust, the brown rust and the stripe rust. The sori are elongated more or less in the direction of the long axis of the leaf. They are quite uniform in size and spacing. Uredospores are round to ovate, spiny, with three to four germ pores and with faintly yellowish orange or nearly colourless walls.

The teleutosori are surrounded by paraphyses. The teleutospores are oblong to cuniform, slightly, constricted at the septum with the apex less thickened and more flattened than that of *P. graminis.* In the Ganges Valley the stripe rust usually appears second to the leaf rust so that the order would be; leaf rust, stripe rust and stem rust. Sometimes this order is not observed and the stripe rust may appear at the same time as the leaf rust. This occurred at Allahabad in 1940 when the stripe rust was in epidemic form as early as the leaf rust but both were late in appearing. It is held that temperature and light intensity as well as the duration of the period of light are factors which play a part in the development of the spore forms from season to season.

The organism. Puccinia glumarum (Schm.) Erikss. Commonly called yellow or stripe rust. Because of the possible confusion of the names yellow and orange rusts Humphrey et. al. (296) proposed that stripe rust be the common name.

As previously mentioned, the uredosori are elongated in the direction of the long axis of the leaf and stem and of a yellow colour. They are arranged in linear rows between the veins and in the direction of the long axis of the leaf which is the easiest differentiating character. The uredospores are nearly round, possess nearly colourless walls, which are covered with fine spines, and measure 23-35 by 20-35 microns. They possess from 6 to 10 germ pores. The telia are longer and more slender than the uredinia and are covered by the epidermis.

Hungerford et al., comparing the forms on wheat and barley, came to the conclusion that they are different and that the names *Puccinia glumarum tritici* and *P. glumarum hordei* were correct. They tested the two forms on 163 named varieties and strains of wheat and found distinct differences between the forms. Mehta (452) reports that there are 9 physiologic forms of the yellow rust in India.

Ferraris (223) states that the stripe rust is found in drier regions than either the leaf rust or stem rust. Sibilia found that temperatures of 35 to 37 degrees Centigrade would completely inhibit the germination of *Puccinia graminis* but only partially inhibit the germination of *P. glumarum*. This may explain the observations of Ferraris. Dickson (190) notes the fact that the uredospores and mycelium of *P. glumarum* are active at very low temperatures which indicate that the rust has a wider range of environmental conditions which it will tolerate than the stem rust.

Life cycle. The life cycle is incompletely known as no alternate host is known nor has the aecial stage been found. Uredospores have been shown to live over the cold season, where one exists, and in such regions they are the only known means of perpetuation. Mehta (452) states that the yellow rust survives on self sown plants in the high altitudes so that there is always a source of infection. After a period of dormancy the uredosori break out and the early plants are the most severely infected. Observations at Allahabad support this statement for the early maturing varieties of wheat, local and Cawnpore 13, are the most severely infected, whereas the late maturing varieties, P. 52, P. 54 and P. 165

are either lightly infected or remain free. P. 4 and P. 111 are intermediate in maturity and amount of infection. It is probably true that other factors, than date of maturity, play a part in the amount of infection occurring on the different varieties but the statement is true as far as the amount of infection by stripe rust on these varieties from season to season is concerned.

Control. Stripe rust is usually a minor disease of wheat on the plains of India. Occasionally it will occur in epidemic form as in the year 1940. The only hope of control that the farmer has is that of resistant varieties.

Bunt or Stinking Smut of Wheat

Host plants. It is found on species of *Triticum*, *Secale* and *Lolium*.

Geographic distribution. It is world-wide.

Appearance on the host plant. There is little evidence of the fungus on the plant until the kernels are examined. It is then found that they are not true kernels but a mass of smut inclosed within the old ovary wall. In some cases the infected plants ripen more rapidly than the normal ones. In some cases there are lighter coloured areas on the leaves of the smutted plants which aid in their identification. In some varieties, as the club wheats, the heads are narrower and there is a tendency to be more straight in the diseased than the normal ones which droop when mature due to the weight of grain.

The organism. Two organisms are usually discussed in connection with the bunt of wheat.

Tilletia caries (D. C.) Tul. is commonly referred to as *T. tritici* (Bjerk.) Wint. in the U. S.

Tilletia levis. Kuhn is commonly referred to as *T. foetans* (Berk. and Curt.) Trel. in the U. S.

As the names appear in literature, and all are in use, it will be well for the student to become familiar with them. About the only important morphological difference between the two species (possibly physiologic forms) is in the spore character. Those of *Tilletia tritici* (*T. caries*) are rough whereas those of *Tilletia levis* (*T. foetans*) are smooth. It is of interest to note that in the United States they are in different areas. The rough-spored *Tilletia tritici* being common on the Pacific coast whereas the smooth-spored *T. levis* is common in regions east of the Rocky mountains. In India Mitra (496) states that *T. caries and T. foetans.* (*T. tritici* and *T. levis*) are more common on the cooler regions of the highlands. *T. tritici* spores measure some 15 to 20 microns in size whereas *T. levis* spores are from 16 to 25 microns in size.

The spores germinate in water or moist air and produce a stout non-septate promycelium which produces a whorl of sporidia at the tip. Dastur (149) found that the number of sporidia is typically 8 and that conjugation is followed by the union of nuclei. Each diploid sporidium then germinates to produce a second shorter and slightly circle-shaped sporidium which produces the infection hyphae.

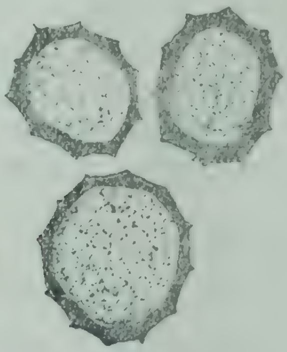


Diagram of spores of bunt of wheat.

Note the thick walls and the small spines. The centres are dark and granular when seen under the oil immersion.

Infection takes place only in the very young plants, the susceptible period being only about 8 to 10 days. Bunt spores grow best at a temperature of 45 to 65°F. Wheat grows best at a temperature of about 75 to 80°F. If the temperature is too high for the smut, but within the optimum for the wheat, it may grow away from the fungus. After infection the smut mycelium grows along with the growing point of the wheat plant and outwardly appears to do no harm. By flowering time the fungus will have accumulated in the ovary and the whole kernel will be a mass of spores instead of living tissue.

The spores may retain their vitality under dry conditions for a long time. Butler (93) states that in mass they may remain viable for as long as 8 years and when separate they will remain viable for 3 years. If damp they do not retain their ability to germinate for more than a few months. Cold and heat together with high humidity are thought to be strong controlling factors so that in India soil infection is probably rare. Most of the infection is from seed-spores.

One of the characteristic things about bunted grain is the odour of fish oil. This is due to trimethylamine which is the substance which gives fish oil its characteristic odour. That this is an inherited character is suggested by the work of Hanna (262) in which he found that some strains of *T. tritici* (*T. caries*) do not have the odour.

Physiologic strains exist in *Tilletia*. In fact *T. tritici* and *T. levis* may be considered to be physiologic forms themselves as they are known to hybridize readily yielding fertile hybrids. Gaines (241) found 5 physiologic forms of bunt of German origin and 4 of American origin. At Pullman, Washington 11 physiologic forms of *T. tritici* and 10 of *T. levis* were indentified. Holton (246) found that hybrids of *T. tritici* and *T. levis* are capable of attacking varieties of wheat that neither parent could attack, thus showing that the hybrid was a new form. This is further evidence of the origin by hybridization of new physiologic forms and perhaps new species.

The smut is also heterothallic. Flor (228) and Hanna (259) have shown that there are sex groups in the smuts. Hanna found that monosporidial cultures failed to produce smut sori whereas paired cultures did. He found the sex groups produced in equal numbers. Flor (228) found that sporidia of a sex group of one smut may unite with sporidia of another sex group of another smut and in this way hybrids are produced.

The life cycle is simple: sporidia to infection hyphae and host infection with the resulting sori and chlamydospores which germinate to produce sporidia again. The production of the seedling infection.

Control. Lee (372) found that copper carbonate gave the best control of bunt of wheat in Victoria. Neil (542) has said the same thing of the control of bunt in New Zealand. He used 2 to 4 ounces per bushel of seed. Padwick (567) observed that where susceptible varieties had not been grown for 2 years the disease was greatly reduced. Resistant varieties also offer a hope for the control of the smut.

Note—An interesting observation was reported by Harris (266) who tested 13 cases of respiratory allergy in Ohio, U. S. A. and found them sensitive to *Tilletia tritici* and *T. levis* as well as to *Ustilago avenae, U. levis, U. zeae* and *U. koleri.* It may be that smut is the cause of some forms of hay fever.

Tilletia Indica Bunt of Wheat

Host plants. The same as for the two previously discussed. i.e., the Species of *Triticum.* So far it has not been reported on *Secale* and *Lolium.*

Geographical distribution. So far it has not been reported outside of India. Mitra (493) states that it has been found on the plains more than the other parts.

Appearance on the host plants. In appearance there is no difference between this bunt and that caused by the other species of Tilletia.

The organism. *Tilletia indica* Mitra.

Mundkur (520) after a study of the number of primary sporidia produced by the smut concluded that as the number is from 32 to 128 it does not belong in *Tilletia* but more properly should be called *Neovossia indica.* (Mitra) Mundkur. The conidia (sporidia) of this smut have not been observed to conjugate which is another characteristic of *Neovossia.* The sporidia of *Tilletia* conjugate while still on the basidium. However, for the present it is probably safer to use the name *Tilletia* but keeping in mind that it may be a *Neovossia.*

The life cycle and mode of infection are essentially the same as for *Tilletia tritici* and *T. levis.* The presence of trimethylamine is also a distinctive characteristic. Infection is usually confined to a few spikelets. Mitra (493) found 3 physiologic forms in India. From what is known of the other species of smuts it would be expected that *Tilletia indica* would be heterothallic.

Control. Mitra (494) found that copper carbonate would control the smut. He also secured good results with uspulun and granosan.

Loose Smut of Wheat

Host Plants. The cultivated species of *Triticum* are the most important host plants.

Geographic distribution. World-wide.

Appearance on the host plant. As indicated in the name, glumes and kernel are destroyed, only the naked rachis remaining. Before the head emerges from the boot it is dark brown with a thin silvery membrane over the spore mass (sorus) which soon ruptures to liberate the spores and thus permit them to be blown about over the field and other plants. This leaves only the bare rachis.

The organism. *Ustilago tritici* (pers.) Rostr.

The smut has been called by a number of names, but the one given above is the accepted one at this time. Cunningham (144) united *Ustilago tritici* and *U. nuda* on wheat as being morphologically indistinguishable.

The spores of *U. tritici* are pale olive-brown but lighter coloured on one side, nearly round, measure some 5 to 10 microns in diameter and bear minute spines on the walls. They germinate to form a promycelium which branches and becomes septate, producing sporidia. Fusion of the sporidia or branches of the promycelium occurs, forming the diploid mycelium. These fused cells (diploid) are capable of producing infection but single cells (haploid) are not capable of doing so.

The common path of entrance into the ovary is through the stigma. A spore blown from the open sorus, at blossoming time of the wheat, falls upon the stigma of the open flower. It germinates quickly sending a thread of mycelium into the ovary where it is established as the grain matures. The fungus remains dormant with the seed until the following season when it is activated by the germinating seed. The fungus follows the growing point of the plant until blossoming time when it rapidly increases in mass within the floral parts and by the time a head emerges from the boot it has completely replaced the grain and destroyed the floral parts.

Control. As in the case of all smuts, which are carried in the seed, surface sterilization is ineffective. The hot water treatment is the method which is considered most successful at this time (see under loose smut of barley. Tapke (777) found that dipping for 95 minutes in water at 120° F. gave good germination and controlled the smut. But he found that in any case it was necessary to increase the seed rate to compensate for the smutted grain and that damaged by the treatment.

There is a wide range of susceptibility and resis-

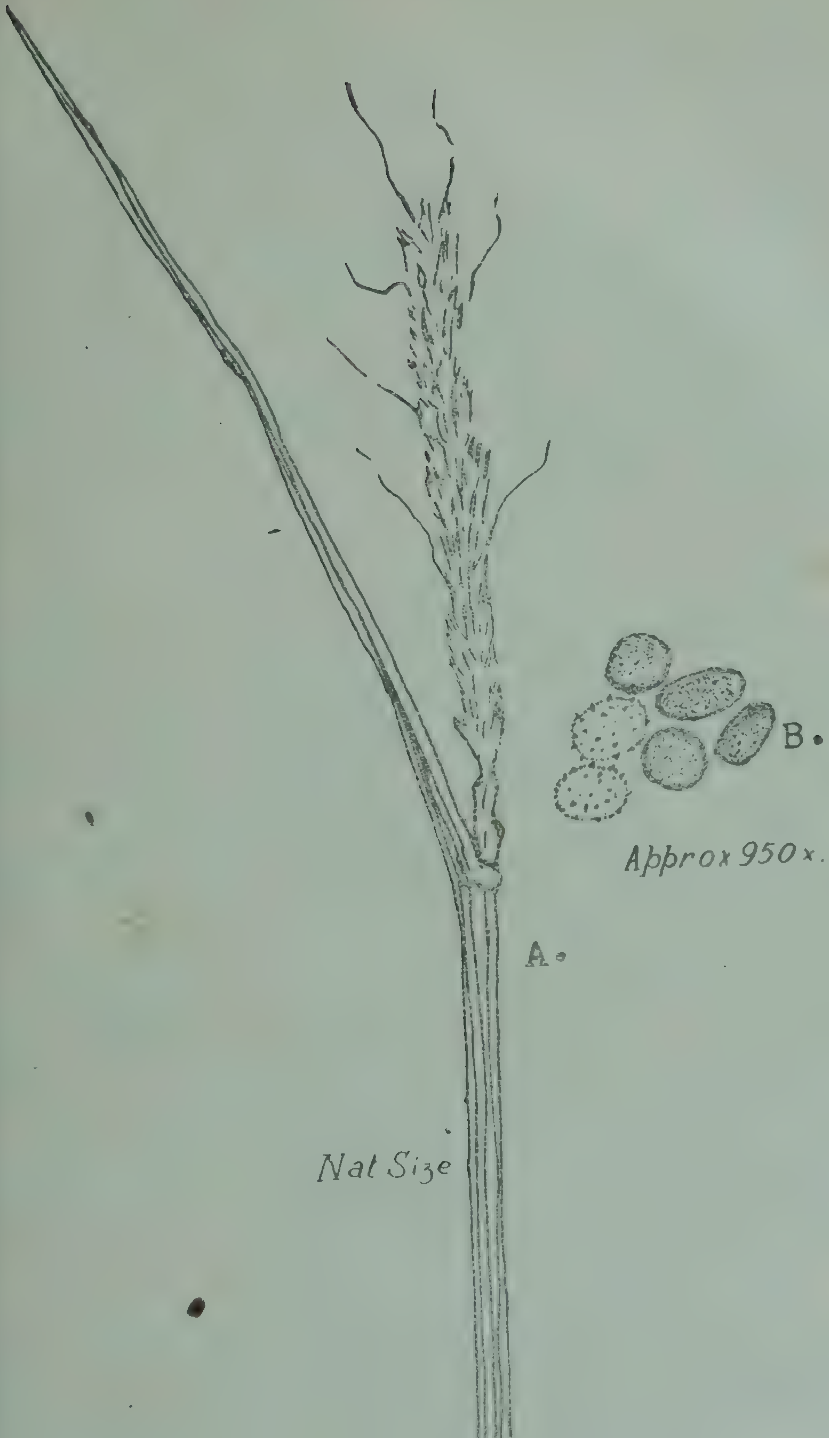


Diagram illustrating loose smut of wheat.

A. Sketch of the smutted spike.

B. Spores. Light coloured spores illustrate the spiny condition. Dark spores illustrate colour.

tance among wheat varieties. The durum wheats have shown more resistance than the vulgare varieties. Of the Indian varieties, P. 114 and P. 165 have shown complete immunity to the loose smut whereas Punjab 591 is very susceptible.

A number of physiologic forms exists. Rodenhiser (661) reported at least 14 physiologic forms in 1928. The existence of so many forms of the smut makes the breeder's problem a difficult one.

The Flag Smut of Wheat

Host Plants. The cultivated varieties of wheat.

Geographical distribution. It is known in India, Japan, Australia, China, Southern Europe, South Africa and the central portion of the United States.

Appearances on the host plant. From the name 'flag smut' one would expect to find it occurring principally on the leaves and especially in the upper ones. However, it may also occur on other parts of the plant including the stems and occasionally on the ears. If a plant is attacked, as a rule, all of the stools will be attacked, and if this occurs no grain is produced. The upper leaves of the diseased plants will be twisted which may be used as an indication of the disease. If grain is formed on such plants it is of little value.

The sori are narrow linear stripes which replace the mesophyll and parenchyma tissue of the leaf. At first they are covered but later they rupture and thus expose the spore masses.

The organism. Urocystis tritici K[illegible]ke.

The most characteristic thing about the flag smut is the spore ball which, as mentioned above, consists of a dark fertile centre and a lighter outer ring of sterile cells. These spore balls are irregularly oblong or subspherical in shape. The spores germinate in place to produce 3 to 4 septate basidia, sometimes non-septate, which produce 3 to 4 basidiospores at the tip. The sporidia are oblong, hyaline, uniseptate at first, but become 1 to 2 septate as they mature and measure 12 to 15 by 3 microns. The sporidia germinate to produce a long slender infection hypha. So far no secondary sporidia have been observed. Infection may occur through the primary shoot or through the young buds of the secondary shoots.

Under ordinary conditions the spores remain viable for a long time. They are carried over on the seed and in the soil as well as on old refuse left over from the previous crop.

Control. Seed treatment. Copper carbonate offers a good seed treatment for the control of flag smut. Resistant varieties are also being used with success. In the United States, by the use of resistant varieties, the disease has been eliminated from some of the growing areas. Yu (942) and his co-workers in China have found wheat varieties resistant to flag smut. Yu has also reported five physiologic forms among the smuts there.

Helminthosporium Leaf Spot of Wheat

Host Plants. Cultivated varieties of wheat.

Geographic distribution. Reported in the northern part of India.

Appearance on the host plant. Mitra (490) states that the symptoms appear in the early stages of the growth of the wheat seedlings. Small (1-2 mm.) yellowish, oval to oblong spots appear on the leaves and leaf sheaths. These spots gradually increase in size and when mature are light brown to dark brown in colour. In general oval or fusiform in shape but may become irregular due to the fusion of two or more into one. The central portion of the spots are irregular in shape, range in colour from straw to grayish brown and bear conidiophores and conidia. A yellowish zone surrounds the diseased area. In general they are very similar to the spots caused by the *Helminthosporium sativum* fungus.

Organism. Helminthosporium tritici repentis Died. The conidiophores are olive to dark olivaceous in colour and emerge through the stomata either singly or in groups. They are 3-9 septate and measure 38.5-300 microns long by 6-11 microns in diameter. The basal cell is slightly enlarged. Knee joints, or geniculations, occur where conidia are borne and fall. The conidia are produced singly, are typically subhyaline, cylindrical, 2-11 septate, slightly constricted at the septa. The basal portion tapers slightly but the distal portion is hemispherical. They vary in size from 45-201.5 microns long by 13-22 microns wide.

Powdery Mildew of Wheat

Host. The main hosts of this mildew are the species of *Triticum.*

Geographic distribution. Wherever wheat is grown this disease is found. It is of minor importance on wheat in India but it sometimes is found associated with a leaf spot of wheat caused by *Septoria tritici* Desm. and when this occurs damage may result.

Appearance on the host plant. It develops on the leaf first as small local spots that have a grayish appearance. They may also be found on leaf sheath and floral structures. As the spots become older they become darker in colour and the portion of the plant below the infected area becomes yellow. The early stages often have a floury appearance from the conidia which cover the surface. The perithecia appear on the surface of the dead or dying leaves and are small and dark.

The organism. Erysiphe graminis tritici Marchal.

The mycelium consists of superficial, sparingly branched, thick-walled hyphae, 4-5 microns wide, which are more or less interwoven so that a web is formed over the surface of the host plant. The short conidiophores rise from the mycelium and bear chains of conidia which are elliptic, hyaline, single-celled bodies that measure 25-30×8-10 microns. They are readily detached from the conidiophores and soon scatter to other plants.

The perithecia appear about the time the plants blossom and fruit. Each perithecium is the result of the union of gametes and when mature is about 200 microns in diameter and globose. These perithecia are scattered over the surface of the host tissues. The appendages are rudimentary and rather few, being the same colour as the perithecia. Asci number from 9 to 30, varying from cylindric to oblong with a rather long pedical. They measure 70-108×25-40 microns. They are typically eight-spored, the spores measuring 20-23× 10-13 microns.

Life cycle. The asci discharge their spores when the wheat plants are small and infection can take place at once. Thus, in India, the infection would probably take place in the later part of December or in January. Conidia soon appear on the diseased spots and the disease spreads rapidly if conditions are optimum.

Control. Use of sprays or dusts has not proved satisfactory. For the most part the investigations have been in the direction of new varieties or hybrids. In 1926 Bifin (61) reported on work done at Cambridge in which hybridization between Persian and Rivet Wheat offered some promise, but on the whole had not been satisfactory. Mains (415), working at the Indiana Station, U. S. A., tested a number of varieties of *Triticum vulgare* and found some resistant to the powdery mildew. He found Axminister (C. I. 1839) and Norka (C. I. 4377) together with several other varieties resistant. The club wheats, *Triticum compactum,* are almost all very susceptible. The spelt wheats, *T. spelta,* and the durum wheats, *T. durum,* were susceptible as were also the poulards, *T. turgidum,* and the Polish wheats, *T. polonisum.* The emmer wheats, *T. diccocum,* and the einkhorn wheats, *T. monococcum, T. dicoccoides, T. persicum* and *T. timophevii* were resistant. He found that C. I. 1839 and C. I. 4377 were resistant to physiologic form No. 1 but not to physiologic form No. 2.

Johnson (315) found that resistance to leaf rust, *Puccinia triticina,* and to powdery mildew were linked in the case of C. I. 4994. Mains (415) found that Norka (C. I. 4377), which is highly resistant to Physiologic form No. 1 of powdery mildew, when crossed with a susceptible variety and the progeny segregated in the F_2 generation, gave evidence that a single factor difference existed and the ratio was 3 resistant to 1 susceptible. This was also true of the linked factors which affected the resistance to *Puccinia triticina.*

Corner (131) studied the resistance to infection by powdery mildew by the resistant variety Norka. He found that germinations and initial penetration were the same as for the susceptible varieties but that the papilla soon died and rarely did haustoria develop. In the case of highly resistant wheat, germination of conidia took place but no infection resulted from the papilla formation.

The above extracts from the work of the various plant breeders is offered to show what is being done and it is left to the thinking student as to what the future may offer in the way of control by resistant varieties.

Sclerotinia Sclerotiorum (Lib.) Mass.

This species was formerly known as S. *libertiana* Fel. but, according to Wakefield (914), should be called S. *sclerotiorum* (Lib.) Massee.

Host plants. As mentioned above, it has a wide range of host plants, many of which are of economic importance and widely cultivated in India. Up to the present time, however, it may be considered among the minor diseases on these crops. Among the host plants of economic importance in India are: *Brassica campestris* var. *sarson; Avena sativa; Cannabis sativa; Cicer arietinum Hordeum vulgare; Lathyrus sativus; Linum* *usitatissimum; Pisum sativum; Triticum vulgare; Vicia hirsuta; Zea maize* and others.

Geographic distribution. The fungus appears to be widely distributed in many countries.

Appearance on the host plant. The symptoms are variable and appear to be subject to the influence of the environment in which the host plant is growing. On the leaves the infections appear as small brown areas on both sides; they may spread to involve the entire leaflet. If the leaflets are heavily infected they may fall to the ground and then develop the heavy saprophytic infection which is also a stage of the fungus. A crown rot may also be evident in fields of alfalfa, clover, peas and vetch. Mundkur (509) reports a boll disease of *Hibiscus sadariffa* due to a *Sclerotinia* which he indentified as *S. sclerotiorum.*

The organism. Sclerotinia sclerotiorum (Lib.) Massee. Although the sexual stage of the fungus has been reported to be a *Botrytis*, most of the workers are of the opinion that it has not been definitely shown to be so and consequently should not be considered such at this time.

The mycelium is much-branched, septate and rich in protoplasm. On the outside of the host tissue it acts as an infection agent when it comes in contact with a portion of the plant which is healthy.

When the food supply is exhausted and the vegetative growth has ceased, the mycelium becomes very dense in spots and within these spots are formed the typical sclerotia. At first these are pink and later they turn black and become smooth. They form on leaves and within the tissues of such plants as carrot and lettuce.

The sclerotia can germinate at once or after a resting period. In some cases they will remain dormant for several years. Mundkur (509) found that the sclerotia were incapable of causing infection unless they were cut, as in the case of the *Sclerotinia sclerotiorum* which he isolated from *Hibiscus*. That is, they could not cause infection without producing apothecia and ascospores. After they were injured they were able to produce infection in contact with the host tissue just as the mycelium does.

When the sclerotia germinate they send up small sprouts which can attain the length of some 5 centimeters. In the light, or upon the surface of the sprout being exposed to air, the sclerotium thickens and becomes flattened, developing into an apothecium which is cup-shaped. The asci are cylindric, 130—135×8—10 microns. The spores are ellipsoid, 9—13×4—6.5 microns and minutely guttulate (with tiny drops). The ascospores and mycelium are both short-lived and the fungus can travel but a short distance in the soil. As mentioned above, no conidia have been found although Mundkur (509) mentions the formation of microconidia on very old cultures of the one from *Hibiscus*.

Control. In many cases the use of sprays does not seem practical for the control of the Sclerotinia diseases. In the case of the one on roselle, Mundkur (509) recommends hand picking of the sclerotia and deep ploughing. Bauer and Huber (49) found the use of calcium cynamid in a dust form to be effective in protecting against S. *fruticola* on blossoms of stone fruits. It is possible that it may be equally effective in such crops as lettuce. But for the field crops it has not been considered economical to use sprays.

At this time it would appear that the best control would be rotation of crops and the destruction of sclerotia together with deep ploughing and resistant varieties.

The Septoria Disease of Wheat

Hosts. Found on wheat and some other grasses.

Geographic distribution. General where wheat is grown.

Appearance on the host plant. Early symptoms are a mottling of the green in more or less circular areas over the blade and sheath. Black pycnidia appear in these areas and are the best means of identification. Heavily infected leaves die prematurely and the new leaves are attacked as they appear.

Luthra and Sattar (396) studied the two common types of *Septoria* on wheat and they report the symptoms of each are quite similar except that in the case of *Septoria tritici,* infections have not been found on the stems and awns, whereas S. *nodorum* is found on the nodes and base of the glumes and stem.

Spots of S. *nodorum* are small, irregular, chocolate brown in colour and dotted with black pycnidia. They are likely to occur on the lower half of the glumes, the nodes or stem.

Spots of S. *tritici* are more or less linear and longitudinally placed on the leaves. They do not occur on stem or nodes.

The organism. *Septoria tritici* Rob. & Desm.
Septoria nodorum Berk.

Septoria tritici spots are larger than those of S. *nodorum,* of a rust yellow in the beginning, later becoming whitish and sometimes with a dark purple border. The pycnidia are immersed, small, subglobose and open by a round pore. Conidia, (pycnospores), are cylindrical, gently curved and with numerous oil droplets. They measure 60-65 × 3.5-5 microns, with 3-5 septa.

Septoria nodorum forms distinct spots on the glumes or nodes only. They are more or less round with a brown border. Pycnidia are gathered on the nodes or glumes and measure 70-100 microns in diameter. The spores are cylindrical, oblong and may be straight or slightly bent. Typically 3-septate when mature, they are colourless when viewed singly but display a pinkish tinge when seen in mass.

Control. Not well worked out. Clean seed and destruction of the old *bhusa* in the infected fields. Crop rotation for one or more years will also aid.

Seedling Blight of Wheat

Hosts. Wide range of hosts as more than one organism is concerned with the blighting.

Geographic distribution. World-wide.

Appearance on the host plant. The first symptoms of the blight are a fading of the colour in the tips of the lower leaves. This is first seen when the seedlings are about four or five inches high. The leaves become gray and then brown. The loss of colour and necrosis proceed rapidly and within a day or so the plants are dead. They will usually be found dead in groups depending upon the condition of the soil. Examination of the dead, or dying, plants will show many dead roots and many with blackened areas. From these areas cultures of at least two, and probably four, fungi can be secured. Of course it does not mean that a pure culture will be secured in each trial but that they may be secured. Losses up to 25 per cent or even more may occur in the low lying areas of the fields.

The organisms. Among the organisms isolated from the diseased roots and stems of diseased wheat seedling are:

Rhizoctonia solani Kuhn.

Helminthosporium sativum Pamm, King and Bakke.

Fusarium species.

Pythium species.

See under Root Rot Diseases.

The Pythium has not as yet been identified but appears to resemble *P. gramnicolum* more than any others. A culture was sent to Dr. R. K. Saksena of

Allahabad University, who has kindly undertaken the identity.

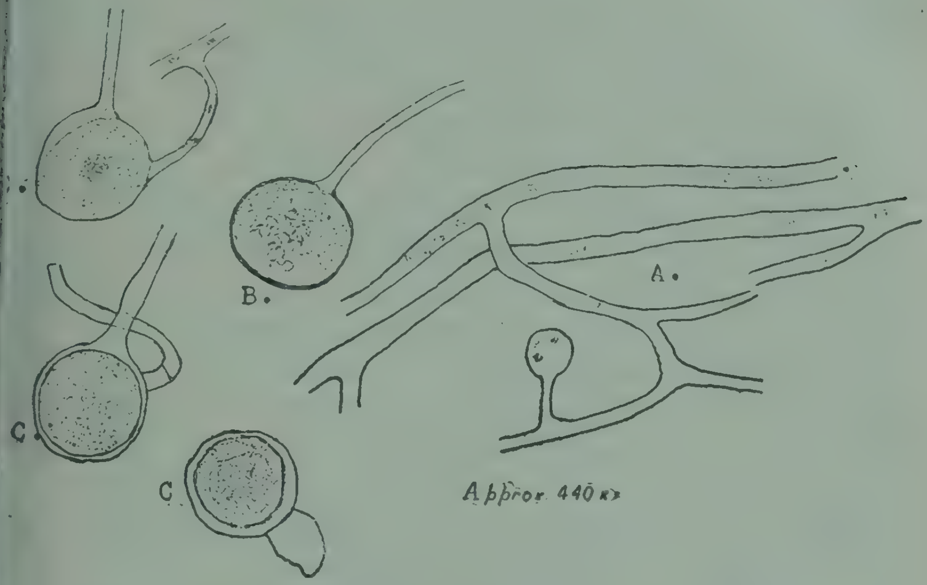


Diagram of a portion of the mycelium, oogonia antheridia and a sporangium of a Pythium isolated from diseased wheat seedlings found on the Allahabad Agricultural Institute Farm 1947.

A. Portion of the mycelium.
B. A sporangium.
C. Antheridia and oogonia.

Using sterile soil and pure cultures of the fungus it was found that the isolation of *Pythium* and *Rhizoctonia* were much more active when taken together than when alone. Pythium alone infected 10 per cent of the seedlings. Rhizoctonia alone infected 6 per cent. When mixed in equal parts the amount of infection was 25 per cent and this corresponded to the percentage of infection observed in the field.

Control. Seed treatment with a fungicide and good drainage for the soil are probably the best known control measures. At this time little is known about the resistance to the disease.

Take-All Disease of Wheat

Hosts. Reported in New York in 1920 on wheat, it has since been found to be able to attack a large number of host plants. Kirby (338) listed some 85 different host plants for the fungus. A large number of wild grasses are also hosts.

Geographic distribution. Widely distributed through the wheat-growing regions of the world.

Appearance on the host plant. The fungus attacks the roots of plants, causing them to become brittle and easily broken. The mycelium grows abundantly about the crown, between the inner leaf sheaths and the stem, or between the leaf during the early growth. The mycelial mat consists of dark brown mycelium which adheres to the culm when the leaf sheaths are stripped off. These often run parallel to each other and thus form ribbon-like strands.

Destruction of the root system deprives the plant of its source of water and nutrient elements and thus the tops starve. They turn white and the name "white heads" or "white top" is often given to the disease.

The organism. In 1928, Fitzpatrick (226), after comparing the fungus of "take-all" of wheat in the U. S. with that from England, Italy France and Japan, concluded that the disease collected from the various localities were identical and that it should be called *Ophiobolus ceraceti* (Berk & Br.) Sacc. In 1925, McKinney (432) reviewed the situation regarding the nomenclature of the fungus and, after stating that there was no longer any of the original European material left to make further comparisons with, and, as there was no difference between the fungus causing "take all" of wheat and the one described by Saccardo as O. *graminis*, therefore that name should be retained. Thus the name now being used is *Ophiobolus graminis* Sacc.

The perithecia appear in groups, or singly, in the tissues of the host and, according to McKinney (433) grow best at a temperature of about 12 degrees centigrade. Padwick (565) reports an *Ophiobolus*-like fungus which appeared on P. 12 wheat in India but that no perithecia were produced. This may be a temperature effect. The perithecia are globose or subglobose, 330-500 microns in diameter, narrowing into a more or less cylindrical beak which is also more or less obliquely attached. The asci are numerous, fascicled, elongate. They measure 90-115×10-13 microns. They are rounded at the ends, 8-spored, thin-walled. There are many paraphyses which are thread-like, flexuous and hyaline. The ascospores are hyaline or yellow, curved, somewhat broader in the middle, and tapering gradually towards the ends. They are 60-90 ×3 microns with 5-7 septa.

Control. Jardine (302) observed that climatic conditions had varied effects upon the incidence of take-all in Oregon. Samuel (667) observed that there was a greater amount of the disease if wheat followed grasses in the farm rotation. *Hordeum murinum* and *Festuca bromoides* were hosts in South Australia. McKinney (433) observed that the percentage of infection increased with the increase of moisture. Webb (920) found that the pH range was 3.2-9.6 and that on potato dextrose decoction the optimum temperature was 24 degrees centigrade with 20, 16 and 28 degrees following in the order named. It has been found that O. *graminis* grew in loose soil better than compacted soil. As a control measure the compacting of the soil and use of nonsusceptible varieties has been suggested.

In the absence of any well-tried resistant varities of wheat the farmer must depend upon rotation, which may include oats and perhaps a legume and see that the soil is in such tilth that the plants make the maximum growth and thus are able to partially withstand the disease.

The Root Rot Diseases

As there are several species of *Helminthosporium* which are found associated with the foot rot of wheat they will be discussed under one heading.

Host range. As this is a disease complex, the range is extremely wide and includes a number of cereals such as barley, rye, oats, and many wild grasses as well as wheat. Henry (278) reported the successful inoculation and infection of 98 species of grasses with *Helminthosporium sativum.*

Geographical distribution. World-wide.

Appearance on the host plant. The symptoms of the disease vary with the age of the plant, the most common being that of dark brown lesions occurring on root, sheath and culms. The lower portion of the plants may rot. Diseased plants may show a darker colour during the early stages of growth. The infection may occur on the heads and be seen as brown restricted lesions on the floral envelope. On the kernels the spots are black whence the name 'black point' has been derived.

The organism. Probably the most common fungus associated with the disease is *Helminthosporium sativum* (Pammel) King and Bakke. Mitra (490) states that *H. Halodes* and *H. tritici repentis* have been found on wheat here in India. Mitra also found one fungus different from any of the above which he isolated from wheat at Pusa. McRae (443) also reported *H. sativum* and one unidentified *Helminthosporium* on wheat at Pusa. Dastur (160) reported a species of *Helminthosporium* which he isolated from 'black point' of wheat. But because of the uncertainty regarding the other species only *H. sativum* will be described at this time.

On barley the disease caused by this fungus has been called 'spot blotch'. On wheat it causes 'foot rot'. The conidiophores are able to emerge from the host tissue from stomata or between the cell-walls. They may arise singly or in twos and threes with the basal cell of each swollen. Conidia are more or less curved, thick-walled, 1 to 10 septate, widest at the middle portion and of a dark olive brown colour. They vary from 110 to 150 by 6 to 7 microns in size.

As in the case of stripe and blotch diseases the fungus is seed-borne but may also live over on the old stubble in the field.

Control. Sanitation and crop rotation should be a part of every control program. In some cases the fungus has been shown to be capable of living over on old stubble for two to three years, hence the necessity for sanitation and rotation. Seed treatment with mercury compounds, as for stripe and blotch, plus resistant varieties will also aid in control.

Some of the six-rowed varieties of *Hordeum vulgare* are resistant to the fungus whereas the two-rowed barley are generally susceptible.

THE COMMON DISEASES OF BARLEY

Although barley does not compare with wheat as to amount grown in India, nevertheless it is grown over most of the country and is subject to a number of serious diseases. The most common of the barley diseases are the rusts and smuts. The smuts will be discussed but the stem and stripe rusts will be omitted under barley as they have been discussed under wheat and there is little difference between the forms found on the two crops.

Other diseases of importance on barley are the three leaf diseases, spot blotch, net blotch and stripe, the root rot diseases and powery mildew. These will be discussed in this chapter.

The Dwarf Rust of Barley

Host plants. Butler (93) stated that the dwarf rust takes the place of the orange rust (leaf rust) in the common group of rusts on barley in India. Dickson

(190) states that the leaf rust infects barley in the Mississippi Valley U. S. A.

Geographic distribution. The rust appears wherever the host is grown although it does not appear very often in India.

Appearance on the host plant. The uredosori are small, round, lemon yellow and scattered over the leaf blade and sheath. The teleutosori are small black crusts, more elongated than the uredosori and are on the sheath and stem.

The organism. Puccinia anomala Rostr. It has also been called *Puccinia simplex* (Koren) Erikass, and Henn. and *P. rubigo-vera* var, *simplex* Koren.

The uredosori are small, and, as stated before, yellow, are ovate in shape and contain globose or ovate spores which bear many very fine spines, possess numerous germ pores and measure 23-30 by 22-26 microns.

Teleutosori are subepidermal and more elongated than the uredosori. The teleutospores may be unicellular or 2-celled and possess a smooth brown wall which is thickened at the apex.

It is a heteroecious rust having its aecial stage on the common Star of Bethlehem (*Ornithogalum umbellatum*) and its uredial and telial stages on barley or wheat.

Life cycle. Where the alternate host occurs the life cycle is easy to account for but the alternate host has not been reported in India so that we must account for the persistence of the rust in some other way. It is possible that the non-existence of the alternate host here is one explanation of the rareness of the rust on the plains. Without the alternate host the uredospores are the only source of infection and for the present we will have to consider it as similar to the other rusts whose uredospores survive in the high altitudes.

Control. It is not considered a serious problem and is included here only for information of the student.

Stem rust of barley—See under wheat.
Stripe rust of barley—See under wheat.

Covered Smut of Barley

Host. Common barley.

Geographic distribution. World-wide.

Appearance on the host. Covered smut differs from loose smut in that the glumes do not slough off. In the field the smutted spikes are distinguished from the healthy ones by their lighter colour and in the difference in the appearance of the awns. Awns of smutted spikes do not spread but remain upright. The basal portion of the spikelet becomes dark and the smutted mass shows through the more or less transparent glume. Smutted plants mature earlier than normal ones and this is a means of identification.

The organism. Ustilago hordei (Pers.) K. & S.

The spores are typically lighter coloured on one side. They are spherical, smooth and measure from 5 to 9 microns.

Control. Covered smut of barley, like bunt of wheat, is controlled by the use of seed treatments such as copper carbonate, Ceresan or some of the more recent fungicides.

Loose smut of Barley

Host plants. It has been found attacking all of the cultivated barley species as well as *Hordeum spontaneum.*

Geographic distribution. World-wide.

Appearance on the host plant. One of the most important diagnostic characters of the loose smut of barley is the erectness of the diseased plants. Another is the empty rachis which is easily seen for the spores have been blown over the field and rest of plants. This

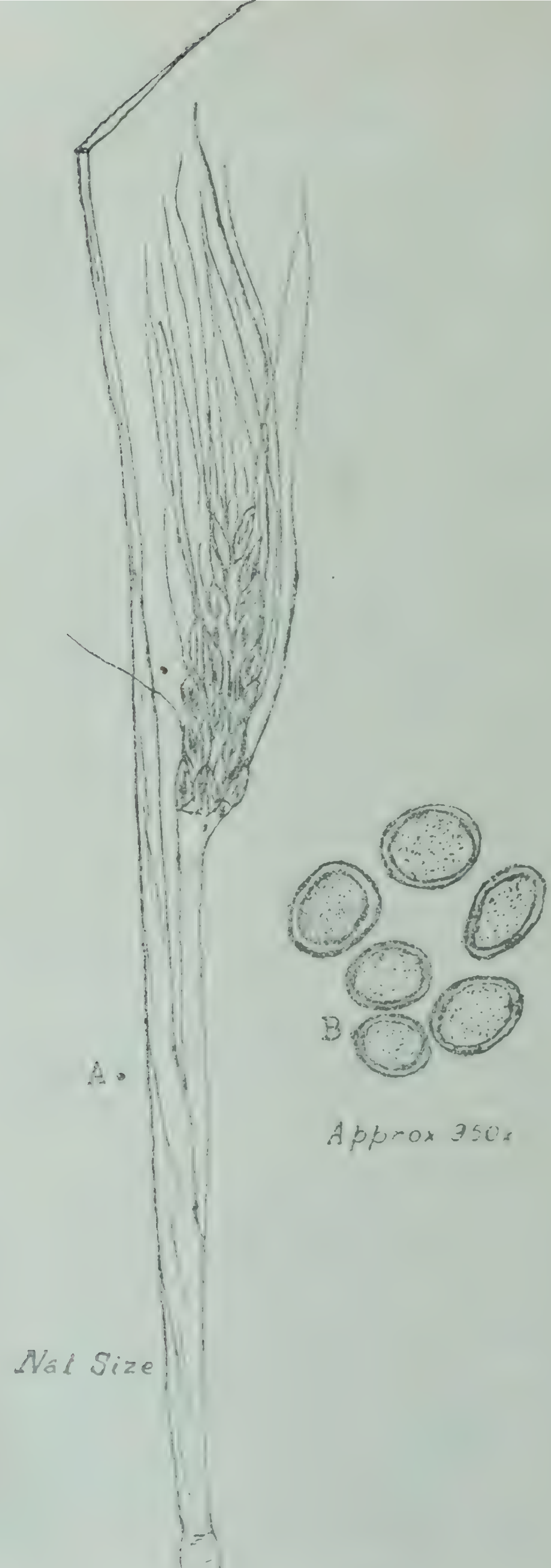


Diagram illustrating covered smut of barley.
A. Smutted spike.
B. Spores.

is a ready means of distinguishing it from the covered smut which is still enclosed within the glumes.

The organism. *Ustilago nuda* (Jens.) K. & S. It has also been called *Ustilago hordei* Rostr. and *U. hordei nuda Jens.*

The fungus is systemic, i.e., throughout the whole plant portion above ground, but the only microscopical evidence of it is to be found in the naked rachis and the erect manner of growth.

The spore mass is olive green-colour which is an aid in distinguishing it from the covered smut. Because of the nature of the sori the spores are widely scattered at flowering time and many fall upon the open flowers of the healthy plants. There on the stigmas they germinate, much as the pollen grains, and the germ tubes pass into the ovaries where they lie dormant until time for the seed to germinate the following season. At that time the activity of the protoplasm of the germinating seed stimulates the fungus hyphae into activity and it grows along with the growing point of the plant and at the end of the season there is only a smutted head where the grain should be.

Thus the cycle is from smutted head and scattered spores to germination in the flower and penetration of the fungus hyphae into the developing seed where it remains dormant until the following season when it begins activity with the seedling.

Control. As the fungus is within the seed, seed treatments with dusts are not effective in controlling the disease. The hot water treatment is the most effective. For barley smut the hot water treatment suggested for covered smut of wheat by Luthra and Sattar (I. J. A. S. IV. pp. 177-199, 1934) is probably best. It is as follows:

1. Presoak in water at 60-80° F. for 4 to 7 hours.
2. Dip in water at 120° F. for 5 minutes.
3. Dip in water at 125-133° F. for 7 minutes.
   Dry quickly.

They have also suggested that the grain may be soaked in water at 105° F. 110° F. and 115° F. for 4, 6 and 8 hours respectively. The disadvantage of these treatments is that they require more apparatus than most of the farmers have.

Mitra and Taslim (496) have suggested a method of treating wheat for control of bunt which should be equally effective for the control of covered smut of barley. They used metal drums which had been painted black to absorb as much of the heat of the sun as possible. In one case the seed was placed in water in the drum and set in the sun from 8 : 00 a.m. until noon on a bright day. The initial temperature at 8 : 00 a.m. was 95° F. and at 12 : 00 N. was 121° F. The seed was removed at 12 : 00 and dried. In another case the drum and water were placed in the sun at 8 : 00 a.m. and 12 : 00 the seed was placed in the water until evening, after which it was removed and dried. Variations of these types were used and all were planted and tested against a control of untreated seed. The smut was completely controlled in the treated seed whereas from 1.50 to 2.00 per cent of smut developed in the check planting. This is not an expensive treatment and any farmer may be able to do it at a minimum of cost.

Resistant varieties offer one of the best methods of controlling the smut. In the United States the Manchuria varieties have been found to be most resistant and from them have been made very good selections. The presence of some 12 physiologic forms of the covered smut of barley makes the breeding for resistant forms a complicated and technical problem.

Spot Blotch of Barley

Geographical Distribution. World-wide. Mitra (400) has given a description of the disease on barley in India.

Appearance on the host plant. Spot blotch on barley seedlings causes a foot and root-rot, a seedling blight, leaf spot, head blight and seed discolouration. The barley plants are stunted, the roots are retarded and lodging increased. Germination of the seed is lowered.

The primary infection is a foot-rot and root-rot of the seedlings. The seedlings appear to be damped off. If severe infection occurs the plants die at the ground level. Many of them die before emergence.

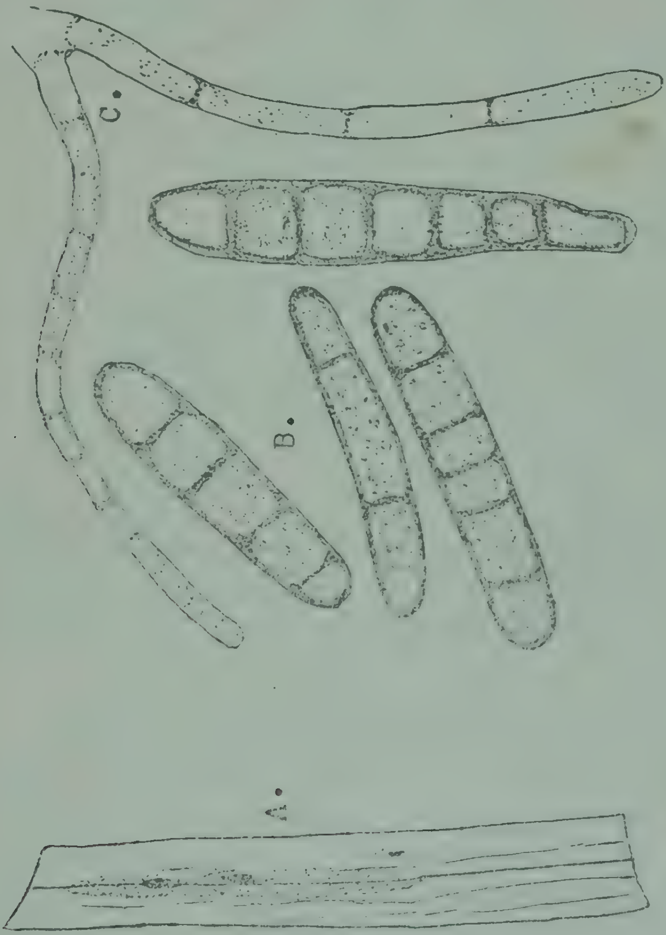


Diagram illustrating leaf spot of barley commonly called spot blotch.

A. Portion of barley leaf showing infected area.

B. Conidia.

C. Conidiophores.

The root tips are killed by local lesions. The later emerging roots may be killed.

Stems attacked at the foot show brownish lesions and soon die. Badly infected seeds do not germinate or the seedlings die very soon after. The diseased plants show very poor root systems. In the United States the growing of barley was practically stopped in the state of Iowa because of the attacks of fungi.

On the leaves the spots are dark-coloured elongate. The centres are likely to be brownish black on which the conidia and conidiophores may be seen with an ordinary hand lens. The margins are lighter in colour.

The organism. Helminthosporium sativum (P.) K. & B.

The mycelium is branched and septate with conidiophores in clusters of two to several. The conidiophores are a fuscous brown in colour, septate, 8-10 microns in width, sometimes swollen between septa. The conidia are solitary, apical, a dark brown in colour, contain from one to seven septa in the cultures locally isolated. (See Figure).

Control. Rotation of crops. At present no resistant strains have been developed with other desirable qualities. Building up the soil fertility with barnyard manures will help control the root rotting phase of the fungus activity.

Late Blight or Net Blotch of Barley.

Host plants. Cultivated varieties of barley and related grasses the world over.

Geographic distribution. It has been recorded in various parts of the world. Butler and Bisby (96) record it from Pusa but Drechsler (199) states that there is some confusion about the identity of the isolation and that it may have been *Helminthosporium sativum*. Mitra (493) states it is restricted to limited areas.

Appearance on the host plant. The infected areas are elongated stripes or lesions which are slightly constricted by the leaf veins. They are dark greenish brown and may be found on leaf blade and sheath. There is no shredding of the diseased areas as in the case of the stripe disease. Where coalescing occurs, the areas become of considerable length but with irregular margins. There is a dark brown reticulated pattern, composed of the diseased areas, which, is so conspicuous that it gave the name 'net blotch' to the disease.

The organism. Helminthosporium teres Sacc.

The perfect stage has been referred to *Pyrenophora teres* (Died). Drechs. It was also referred to *Pleospora teres* Died. Both of these genera are under the family *Pleosporaceae*, of the order *Sphaeriales* of the *Pyrenomycetes* of the *Ascomycetes*.

The fungus is intercellular as in the case of the stripe disease. The conidiophores emerge in clusters from the stomata, are reddish brown, septate and measure up to 180 microns in length. Conidia are borne singly on the blunt tips of the conidiophores. They are mostly straight, 100 to 150 by 15 to 20 microns with from 7 to 14 septa and with the greatest width in the middle. They are greenish gray when young but become gray black when old. New ones may be formed in the same place after the old ones have fallen.

The Stripe Disease of Barley

Host plants. It appears to be confined to the cultivated species of barley.

Geographic distribution. It is world-wide but appears to be more serious in Europe than in India and the U. S. Mitra (493) states it is rare in India.

Appearance on the host plant. The first appearance of the disease is small pale spots on leaves and sheath. The spots differ from those on oats, being longer and may be continuous from sheath to the leaf tip. The margins of the diseased areas dry and become brown. The plants may be short and possess many tillers; or they may be of the other extreme, i.e., long and few tillers. In some cases the ear does not emerge from the boot. Awns of infected heads are usually twisted and grains poorly formed.

The organism. Helminthosporium gramineum Rabenh.

It was formerly called *Heterosporium gramineum* Oudeman. The conidia of *Heterosporium* are echinulate and that prevents this organism from being retained in that genus.

The fungus gains entrance to the host plant primordial tissue and becomes systemic. Examination of the diseased tissues will show the mycelium intercellular and this condition develops before blossoming time. The infected areas develop spores about the time of blossoming and if conditions are favourable for spore germination, floral infection will result, followed by infection of the embryos in which the fungus lies dormant until the seed germinates the following season. Thus the primary infections are the floral infections. Although the perfect stage has been reported to be *Pleospora*, a genus closely related to *Ophiobolus* of the *Ascomycetes*. Dickson (190) considers the evidence too poor to warrant acceptance.

Control. Sanitation or crop rotation will no doubt be best for the farmer, but seed treatment with mercury compounds will also help. Resistant varieties have been shown to be a possibility in the U. S. where a number of varieties (see Dickson (190) are being developed. In India at present there does not appear need of much concern but there is always the possibility of new forms appearing and the plant pathologist needs to be forearmed.

Erysiphe Graminis Hordei Marchal

Hosts. The hosts in this case are varieties of the genus *Hordeum*, both cultivated and wild.

Geographical distribution. Distribution is general in the humid and semi-humid areas of the world, being more severe in the winter barley sections of the United States than in other sections.

Appearance on the host plant. The fungus mycelium is a light gray on the surface of the leaf, leaf sheath and flowers. It darkens with age and numerous perithecia appear over the surface. When the infection is severe the portion of the leaf below the infection turns light brown.

The organism. It was formerly known as *Erysiphe graminis* DC, together with the species occurring on wheat, on oats and on rye. In 1902 and 1903 separate species were set up which made the one occurring on each of the four species of crop plants a separate species in each case adding the latinized name of the genus of the host plant to complete the specific name. Thus the barley mildew is known as *Erysiphe graminis hordei* Marchal.

Life cycle. See *Erysiphe graminis tritici.* It has been reported that the conidia live over winter and that the perithecia play a small part in the life cycle.

Control. Dusts and sprays are of little avail in the case of barley mildew as in the case of other cereal mildews. As in the instance of wheat, it is from resistant varieties that the most help may be expected. In the United States some promising varieties have been developed which have been reported by Mains (413), Tidd (816) and others. In greenhouse trials with 85 varieties of barley in the seedling stage it was found that the reactions indicated resistance was inherited in definite mendelian fashion. Adults have been shown to be more resistant than seedlings.

Root Rots of Barley

The root-rotting fungi which are found on the barley plants are similar to those already mentioned on wheat. It is probably true that rarely, if ever, a plant dies of a single root rot disease. Along with the pathogen, which is primary in the infection, will be a number that are more or less saprophytic and when the plant is weakened by the active parasite the others take advantage of the situation to get in on the kill.

Host. Most of the cereals and many other plants.

Geographic distribution. World-wide.

Appearance on the host plant. Symptoms are rarely so distinct of one type of disease or the other that one can say certainly which one of the possible pathogens is the active one. But in the Allahabad area one can be fairly certain of the pathogen being a species of *Fusarium, a Rhizoctonia, a Sclerotinum* or *a Helminthosporium.*

The first symptoms likely to be observed will be a yellowing of the leaves from the tips downward. Old leaves die and wither. Examination of the stems will usually show them dark-coloured and, in the younger plants, rotting at the ground level. If the plants are pulled from the ground they will be seen to have roots that are blackened and decayed. From these areas one may find any or all of the first three fungi named. The *Helminthosporium* is more likely to be found at the crown or ground level. Foot rot diseases are especially bad in low ground. In the 1946-47 season, barley, on the Institute farm which had been planted on a flooded area, was badly damaged by root rot fungi. *Sclerotium rolfsii* and a *Helminthosporium* species were found in the diseased roots and stems.

The organisms. Helminthosporium sp. In this case it appeared to be very like *H. sativum* (Pammel) King and Bakke. *Rhizoctonia solani.* Kuhn.

Control. Rotation. Organic manure.

Fusarium species..

Sclerotium rolfsii. . Sacc.

The student may ask why all of these are given when it is not possible, except by laboratory methods, to tell which one is most responsible for the trouble. But the answer to that is that all are soil-borne and that the symptoms which have been given above indicate that one or all may be present in the soil about the plants. The same treatments which will aid in the control of one will also aid in the control of the others. After all, the farmer is not so concerned about the particular fungus on his plants as he is how to control it.

Control. Good drainage is one of the most essential things. Organic manures are among the best agents to control the soil-borne diseases. Seed treatment will help but much of the root rotting is after the seed treatment has lost its value. Chester (105) mentions the use of phosphate fertilizers for the building up of the soil.

Some of the six-rowed barleys have been reported as resistant to *Helminthosporium sativum* whereas the two-rowed are mostly susceptible.

THE COMMON OAT DISEASES

The oat is a minor crop in India yet at the same time it is becoming more and more common as a forage crop where a quick growth is desired. It is being used as a catch crop in some rotations.

There are not many diseases which are serious on the oat. Rusts are as yet unreported in India. Smuts include the loose and covered smuts. A *Helminthosporium* leaf spot and the root rot disease are the most of the common diseases reported on the oat in India.

Leaf Spot of Oats.

Host plants. Species of *Avena.*

Geographic distribution. Reported from all oat-growing regions of the world.

Appearance on the host plant. The disease appears on the leaf blade and occasionally on the sheath, as elongated gray brown lesions, limited on either side by the veins so that the long axis of the lesion is parallel to that of the leaf. The central portions of the lesions are usually dark with a grey centre upon which are found the spores.

The organism. Helminthosporium avenae Eidam.

The mycelium is intercellular and although no haustoria are found, the plant tissues are soon killed. The necrotic areas become first light and then dark with the dark centre and gray spot referred to above. Conidiophores emerge from the stomata of the old diseased areas as brown, simple, septate hyphae with rather abrupt bends where the conidia have been formed and fallen off, the conidial scar being visible at the bend. The conidia are formed terminally but as they mature the conidiophore continues to elongate and the conidium is pushed to one side before falling as a mature spore. The conidial scar is often referred to as a knee bend in the conidiophore. The conidia are deep brown, long, cylindrical bodies with thick walls and from four to six septa. They measure 80 to 100 by 15 to 16 microns.

Control. Destruction of all infected plant material, use of clean seed (although it is not considered that seed treatment is very effective) and rotation. As evidence in favour of seed treatment, Carroll (97) using ceresan, agrosan and abavit as seed dusts, found the yields increased about 10 per cent and the disease reduced some 25 per cent.

The Covered Smut of Oats

Host plants. The host for the covered smut is the same as for the loose smut. Simmonds (695) found the wild oat (*Avena fatua*) infected with *Ustilago levis* in Western Canada.

Geographic distribution. It is world-wide in distribution but there may be differences in the races which exist in different parts of the world.

Appearance on the host plant. As stated in the discussion on loose smut, it is sometimes difficult to tell the covered and loose smuts apart in the field. The inclosing membrane is more persistent in the case of the covered smut and the ends of the glumes are likely to be longer and preserved for a longer period. The delicate inclosing membrane is about the only identifying character which can be depended on.

The organism. *Ustilago levis* (K and S) Magn. or *U. kolleri* Wil. Both names are used but the latter appears to be more general in India. In the United States the former is the more common. In European countries it is often referred to as *Ustilago avenae levis.*

The spore mass is black-brown with the spore adhering together at first. According to Mundkur (512) the sori on the leaves are parallel to the veins and produce a longitudinal shredding of the tissue. The spores are smooth and lighter coloured on one side, measuring from 5-9 microns in diameter.

Most of the infections are from the seed-borne spores, although some infections are from the soil-borne spores. The infection takes place in the primordial region of the growing point, as for the loose smut, and the fungus grows upward with the tip of the stem until flowering time when the inflorescence is completely replaced by the fungus and nothing appears in the ear but a mass of black sori.

The suggestion that the loose and covered smuts may be single races of a single species is given support by the work of Holton (286) who was able to show that *Ustilago avenae* and *U. levis* (*U. kolleri*) are infertile and that the hybrids will produce infection on oats. Rodenhiser in 1928 reported 18 specialized forms of *U. avenae* and 5 forms of *U. levis.* Dickson (190) in 1939 stated that there were 17 forms of *U. avenae* and 9 forms of *U. levis.* As a matter of interest it might be well to note that Welch (924) found that plants infected with loose and covered smuts were more susceptible to rust than the smut free plants.

Control. The same control measures as recommended for the loose smut are also effective for the covered smuts. A volatile fungicide, such as formaldehyde, is needed for the control of the covered smut as other fungicides are not able to penetrate in between the husk and the kernel.

For formaldehyde; mix one pint of the 40 per cent commercial brand with one pint of water and apply uniformly over the grain at the rate of 1 quart of the mixture to approximately 20 maunds of grain. Mix the grain well as the spray is being applied, using a clean floor for the operation. Dry well and, if to be sacked, see that the sacks have been sterilized. If to be stored for a time it must be shifted and aerated well before storing as the fumes may destroy the vitality.

Neil (542) reported that carbonate and copper oxychloride were effective in controlling loose and covered smut. Ceresan, among the mercury compounds gave almost perfect control. In addition, it increased the number of plants. Agrosan G gave better control but did not give as good results in the increase in plants.

Padwick (567) reported that oat varieties showing resistance were being developed in New Delhi and Pusa.

An interesting possibility in the control of smuts (see under Biol. Agents Chapt. IV) is the discovery that certain bacteria are antagonistic to the smut fungi. Johnson (316) reports the isolation of a coccus from *Ustilago avenae* that has shown antibiotic properties when asso-with *U. avenae U. levis* and *U. zeae*. At the same time a spore-bearing rod and a *Myxobacterium* were also isolated and found to be antibiotic. We need to know more about this phase of symbiosis and it may be possible to pit organisms against each other and thus be able to effect a measure of control.

The Loose Smut of Oats

Host plants. Cultivated varieties of the genus *Avena* and especially *Avena sativa.*

Geographic distribution. World-wide.

Appearance on the host plant. The fungus attacks the flowers and especially the ovary which is replaced by a mass of spores. In this respect covered and loose smut of oats are very similar. The glumes fall away sooner in the case of loose smut.

The organism. Ustilao avenae (Pers.) Jan.

The sori are covered with protective membrane which soon ruptures and frees the spore mass which is then a loose powdery mass, olive brown in colour and soon scattered over the field and neighbouring plants. The spores are nearly spherical lighter coloured on one side, finely echinulate and from 5 to 9 microns in size. They germinate to form a 4 to 5-celled promycelum with a sporidium at each segment.

As the oat seedlings emerge from the protecting seed coats they are very delicate. At this time the smut chlamydospores germinate to produce promycelia and sporidia and if these contact the young seedling growing point entry is easy and, once inside, the fungus grows along with the host plant in the cells of the embryonic region. This sort of symbiotic relationship goes on until the oat plants have reached maturity and are ready to flower, without any evidence of injury being observable. A section of the stem at this time will not disclose the mycelium of the fungus. It is only by sectioning the growing tip that the smut fungus may be seen. But at flowering time it is observed that there is no flower present but instead a mass of black material in place of the floral organs. These quickly rupture and as the other plants flower the spores from the smutted heads are scattered over them. Some of the spores are scattered over the grains at harvest time and carried to the store rooms. If not disinfected these grains are

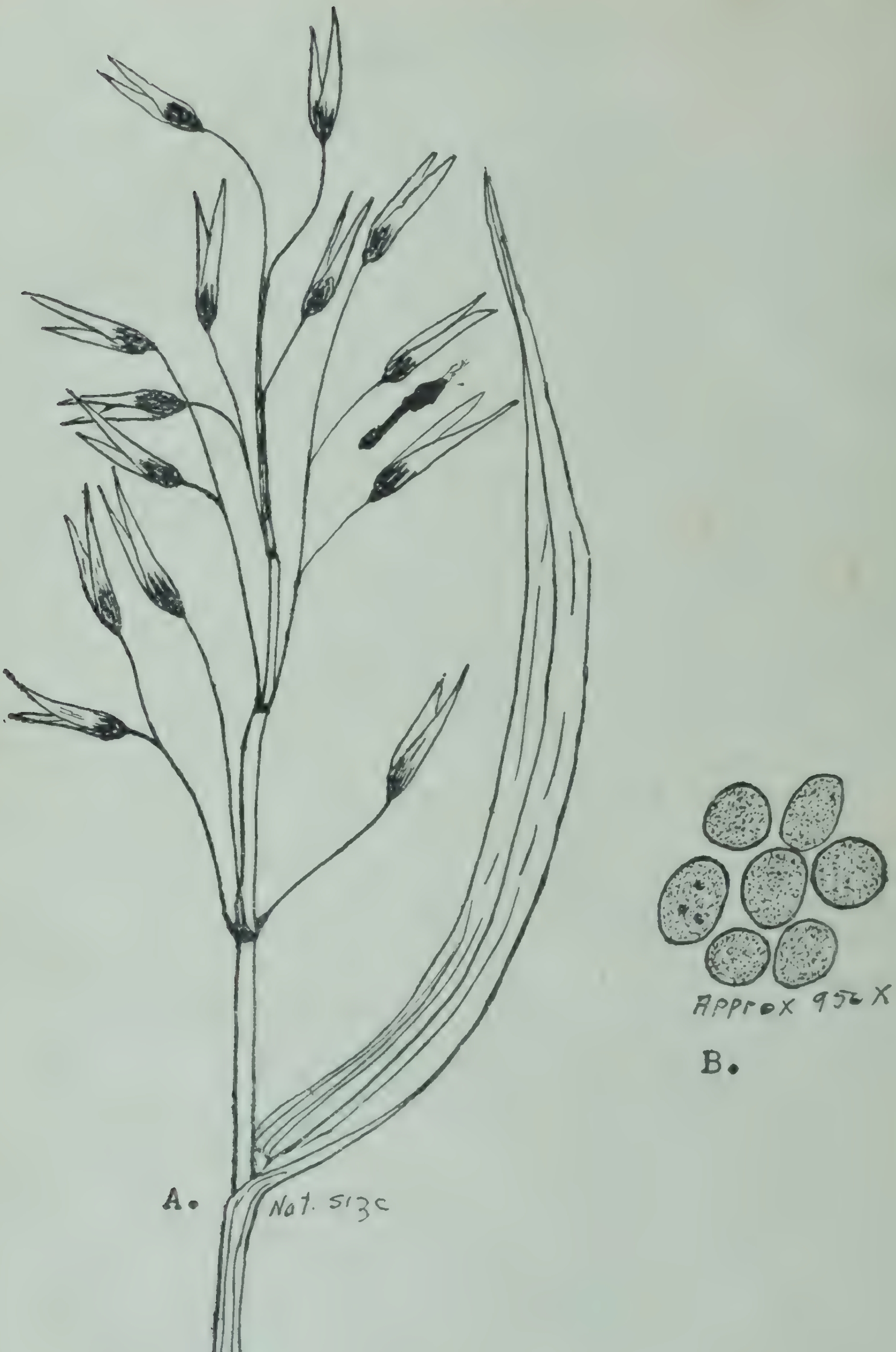


Diagram illustrating the loose smut of oats.
A. Diagram of smutted panicle.
B. Spores.

sources of infection at planting time. Other spores are scattered over the soil, or old refuse on the soil, and thus are a source of infection for the next year.

Thus a life cycle sketch would be: chlamydospore germination at the time of seed germination which is followed by plumule infection and the resulting smutting of the head. The spores are scattered and become the source of infection for the following year.

Control. Seed disinfection is perhaps the most effective method. For the loose smut copper carbonate, the mercury compounds and formalin, 1 part to 320 parts of water as a sprinkle, have proved effective. During recent years copper carbonate has become increasingly popular. Used at the rate of 2 to 3 ounces per bushel it has been very satisfactory.

CHAPTER VII

THE MORE COMMON DISEASES OF JOWAR, BAJRA AND MAIZE IN NORTHERN INDIA

Common Diseases of Jowar.

The jowar crop is one of the very important fodder crops in India and it is also subject to a number of diseases. These include smuts, rust, various leaf spots, downy mildew and root rot diseases. The more common ones will be discussed in the following chapter.

Red leaf spot of Jowar

Host plants. Cultivated jowar and some other grasses. Galloway (238) and Boning and Wallner (76) reported it on maize.

Geographic distribution. It does not appear to have become serious in other parts of the world but is one of the most common of the jowar diseases in India.

Appearance on the host plant. In some ways red leaf spot of jowar is very similar to the red rot of sugarcane. The leaf spots are similar, the reddening of the tissues is similar and the formation of the acervuli is also similar. The fungus of red leaf spot is confined to the diseased areas but penetrates the entire thickness of the leaf. The leaves often break where the spots occur but the broken half does not wither and become brown as in the case of red rot. Infections are confined to the leaves and very few are found on the stems.

The organism. Colletotrichum gramnicolum (Ces.) Wilson.

The formation of acervuli is very similar to that in the case of red rot. The acervuli are more numerous than in red rot and perhaps more prominent. The conidia are hyaline, distinctly crescent-shaped, with one or two oil droplets in the central region. They measure 20—30×4.5 microns. The setae are similar to those of red rot and measure some 175×4—6 microns. They are tapering and lighter coloured toward the tips.

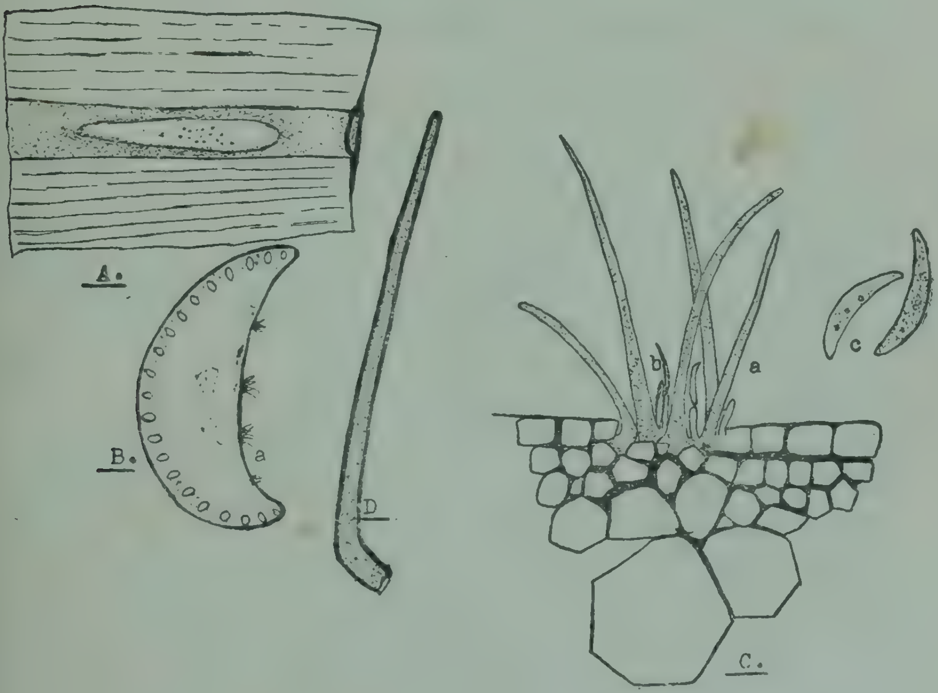


Diagram illustrating red spot on jowar.

A. Section of a leaf showing infected area. Note the area is along the midrib.

B. Cross section of a midrib showing location of acervuli, a. marked by the black setae.

C. Section of a portion of the midrib showing the setae a., conidia b., and enlarged conidia c.

D. Single setum enlarged.

Choudhuri (115) considered the disease on maize and on jowar identical. This article should be read in this connection.

Control. There does not appear to be any recommendation for the control of the disease. It is commonly present in the fields but has not become anything like a limiting factor in the jowar-growing regions.

Boning and Wallner (76) reported the fungus causing a foot rot of maize in Bavaria but there is no evidence of it doing so in India.

Leaf Spot of Andropogon Sorghum

Host plants. Maize, sorghum, Johnson grass and other grasses.

Geographic distribution. It has been reported in the U. S., South Africa and India. Ramakrishnan (637) described it on *Andropogon sorghum* in India.

Appearance on the host plant. The spots have a reddish appearance and are first seen upon the lower leaves. Later the upper leaves may also become infected. The spots are elliptical to round with the long axis of the spots parallel to that of the leaf. In severe cases the whole leaf may become covered so that little green may be left. The spots measure from 5—15 × 3—5 mm. but when coalescing occurs they may become very large. Ramakrishnan states that the colour of the spots is influenced by the colour of the leaves. The darker the leaves the darker the diseased spots.

The organism. Cercospora sorghi E. & E.

The hyphae are intercellular at first, later intracellular, and are principally found in the mesophyll tissue. They are limited in their lateral movement by the lignified vascular bundles.

As in the case of other *Cercospora* the hyphae form preudostromatic masses beneath the epidermis which produce the conidiphores. These are septate, dark brown in colour and average 71.5 by 4.6 microns with the typical knee joints. The conidia are hyaline, multiseptate, broadest at the base and tapering slightly toward the tip.

Ramakrishnan states that infection takes place through the stomata.

Control. Rotation and destruction of diseased plants.

The Downy Mildew of Sorghum

Hosts. Maize, teosinte, sorghum and related grasses.

Geographic distribution. In the more tropical regions where humidity and temperature are high this disease is prevalent. It was reported in the Philippines by Weston (928) and in India by Butler (93). It has since been reported from a number of areas of the maize belt.

Appearance of the host plant. One of the first noticeable effects of the fungus is to cause a dwarfing of the plants. The leaves are usually yellowish in colour with a puckered appearance and bearing streaks of lighter colour. The nodes are often shorter and the plants bear a bunchy topped appearance. On the spike of jowar the floral parts become distorted and resemble narrow leaves which curl and twist, giving a peculiar witches' broom appearance. The name "green ear" is often applied to the floral parts of the infected plants.

The organism. It was first named *Sclerospora indica* by Butler and then in 1936 Uppel and Weston (852) merged it with *S. philippinensis.* Thus it is now known as *Sclerospora philippinensis*, Weston.

The infections occur from the time the plants are in the third leaf stage until they are practically mature. However, most of the infections are during the early stages of the plant growth. The conidiophores, which are large, are dichotomously-branched with a large basal cell. The conidia, borne at the tips of the branches, are ellipsoid to oval, 27—39×17—21 microns. In 1921, Storey and Mclean (J. Ag. Resh. xx, pp. 669-684, 1921) found another *Sclerospora* on maize in the Philippines, which they named *Sclerospora spontanea.* The conidia of this species differ from those described above being long, and slender in form. Conidiophores are produced at night from the stomata and are 150-400 microns long by 15—26 microns wide. Germination of conidia is always by germ tube.

Life cycle. From the above the life cycle is easily worked out. With infections taking place at the third leaf stage and the oospores overwintering, or carrying over the dry season, one has only to follow the growth of the host plant to outline the fungus life cycle.

Control. Since the source of inoculum is the diseased host plants in the neighbourhood, the old diseased material left in the field from the previous season's crop as well as the oospores which may be in the soil, it follows that sanitation and removal of the diseased plants, together with rotation of crops are the most important means of control. Weston (928) recommended seed treatment for *Sclerospora.* See previous discussion.

Loose Smut of Jowar

Host plants. Many of the species of *Sorghum.*

Geographic distribution. World-wide.

Appearance on the host plant. Affected plants are stunted, have thinner culms and there is a greater tendency to tiller.

The organism. Sphacelotheca sorghi (Link) Clinton.

It has also been called *Sorosporium sorghi* Link and *Tilletia sorghi* Tulasne.

The kernel is replaced by a fungus membrane which is composed of long thin cells that are easily broken, permitting the spores to escape. They are round, somewhat irregular, light brown with smooth walls. They measure from 5 to 10 microns in diameter and upon germination produce a typical 4-celled basidium and sporidia. Much that has been said regarding the grain smut will also apply to the loose smut. This is especially true of the physiologic forms. The life cycle of S. *sorghi* is essentially the same as that of S. *cruenta.*

Kulkarni (351) found that the optimum temperature for the germination of the spores of S. *sorghi* lies between 21 and 30°C. This temperature is low for the grain which has an optimum temperature of 30 to 40°C. Where the temperature is optimum for the grain it is too high for the smut so that there is little danger of an epidemic on the plains. This has been shown to be true in the U. S. as well (463). Early planting resulted in heavy smut infections. Medium to low temperatures and medium to low soil moisture resulted in high smut infection but as the temperature rose about 75° F. the infection decreased.

Control. See *Sphacelotheca cruenta* below.

The grain Smut of Jowar

Host plants. It is found on both wild and cultivated species of *Sorghum.*

Geographic distribution. Rather general in Africa and Asia where jowar is grown. Common in Madras, Central Provinces, Bombay, Burma and the Dehra Dun areas. It is known in the United States but is not widely distributed.

Appearance on the host plant. This smut differs from the other smuts described so far in the manner of infection. This one is found attacking individual kernels. Even though all of the kernels of a head may be infected they are infected separately. The infected spikelets may be elongated and hypertrophied in some cases while in others there may be no outward evidence of the smut. The stigma is not included in the sorus and this fact may make the smutted kernel appear so normal that the diseased condition is not observed until harvest time. The floral envelope is usually reddish and this is an aid in finding the smutted kernels.

The organism. Sphacelotheca cruenta (Kuhn) Potter.

The sori are of two kinds according to the manner of growth, large and small. The large sori are mostly fungus tissue with very little plant tissue. The small ones are mostly ovary tissue with only a few spores. The central columella persists after the outer wall has

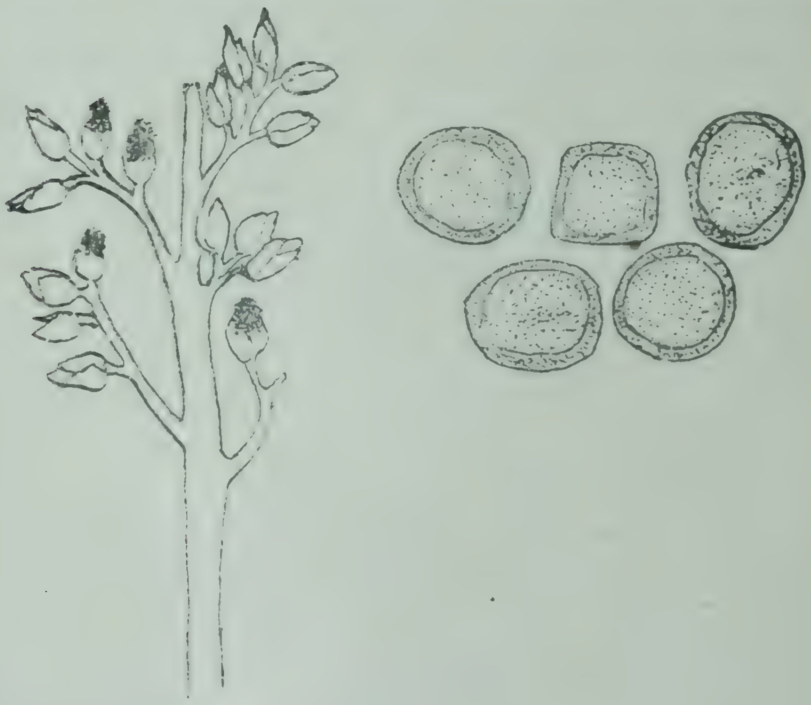


Diagram illustrating the grain smut of jowar.

A. A portion of the panicle of jowar with the smutted kernels inked black.

B. Spores. Note the thick walls and the irregular shapes.

been broken away. The spores are oval, dark brown, when seen in mass, but an olive brown when seen individually. They measure some 5 to 9 microns in diameter and possess smooth walls.

The chlamydospores germinate to produce a 4 celled promycelium and sporidia. Secondary sporidia may also be produced.

Reed (650) found that the percentage of kernel smut of sorghum was negatively correlated with the percentage of moisture. Richie was also of the opinion that climatic conditions played a large part in the percentage of smut (S. *sorghi* and S. *cruenta*) in Tanganyika but he observed that dry weather was a factor in the control which is in agreement with the observations of Reed. It is probable, however, that more work needs to be done in this connection.

Infection takes place in the seedling stage with a systemic development of the fungus and the establishment of the mycelium in the stem primordia. The infected plants are dwarfed and the smutted heads usually appear from the boot before the normal ones do. As these early maturing sori develop the spores are scattered over the mature grain. They are not thought to be able to retain their vitality long under moist conditions. According to Butler (93) they may remain viable much longer when kept dry.

The life cycle is similar to that of the other smuts of sorghum. Spores may be seed borne or soil borne with seedling infection the rule.

Physiologic specialization in *Sphacelotheca* is known through the work of Melchoirs (463), Rodenhiser (660) and others. Rodenhiser (661) demonstrated heterothallism in both S. *sorghi* and S. *cruenta* in 1932. In 1934 (661) he demonstrated that interspecific hybrids were less virulent than intraspecific hybrids. This would tend to indicate that they are closely related and may be but physiologic forms of the same species.

In 1931 Swanson (770) reported that resistance to sorghum kernel smut was a single factor difference with susceptibility dominant. Marcy (420) found a resistance factor R which was carried by dwarf milo and was epistatic to a susceptible factor S carried by susceptible varieties. She also found another factor B for resistance which was hypostatic to S. This indicates some of the complexity met with in the study of the inheritance of resistance to smut in the sorghums.

Control. Anstead (29) in 1924 recommended Germisan, a mercury compound, for the control of *Sphacelotheca sorghi.* Dastur (151) in 1925 recommended copper carbonate at the rate of ½ oz. to 2 ℔s. of jowar seed. In 1926 (154) he recommended ¾ of an oz. of copper carbonate to 6 seers of seed and in 1928 (154) copper carbonate was recommended by Government for S. *sorghi* and S. *cruenta.* Uppal (839) recommended 200 mesh sulphur at the rate of 3 to 4 oz. per 60 ℔s. seed.

Of the varieties of jowar which appear to be most resistant to the two smuts in the Bombay Presidency (839) Dwarf Standard, White Milo, and Spur Feterita have been listed first. On the other hand Blackull, Red and Down Kafie and Shallu are from 15 to 100% susceptible. The first three should be useful in breeding.

The Head Smut of Jowar

Host plants. Sorghum species. Sweet sorghums and grain sorghums are all quite susceptible whereas such as feterita, milo, broom corn, kafir and kaolings are more or less resistant.

Geographic distribution. It is common in various parts of India, the United States of America, Europe and Africa.

Appearance on the host plant. Sori on the leaf blades and in the heads are both prominent with the head sori the more prominent. In some cases these masses of fungus material may reach several inches in length. They may be compact or they may be composed of a number of finger-like growths which are powdery in appearance.

The organism. Sorosporium reilianum (Kuho) McAlpine. This is a physiologic race of the one on maize but cannot infect maize nor can the form on maize infect jowar. Otherwise the two appear identical. Hanna (224) found that *Sorosporium reilianum* is heterothallic and behaves the same as *Ustilago zeae*. As the spore characters and sori characters are the same, as for the form on maize, they will not be repeated here but the student is referred to that section.

Control. As the organism is soil borne it follows that any plan of control must include sanitation and rotation of crops. *Ustilago zeae* and *Sorosporium reilianum* will hybridize so that new forms may appear at any time in an area where the two forms of smut exist. Rotation and seed treatment with one of the recommended seed treatment compounds are aids in control.

The Long Smut of Sorghum

Host plants. Both *Sorghum* and millet species are attacked.

Geographical distribution. Up to the present it appears to be confined to Africa and Asia. In India it is most common in the Madras and Sind regions.

Appearance on the host plant. Only individual flowers are attacked but when this appears the kernel becomes very much enlarged and assumes a curved shape, the sorus later becoming brownish yellow in colour. In the millets the sori are likely to be more pear-shaped and much darker in colour.

The organism. Tolyposporium penicillariae Bref. occurs on millets. *Tolyposporium ehrenbergii* (Kuehn) Pat.

The spores are held in more or less permanent spore balls in the sori by a mucilaginous material and also by a surrounding membrane. They do not separate at germination time but germinate in place. The promycelium is typically 4-celled to begin with but may branch and form numerous sporidia which are either single or in chains. The sporidia germinate readily in distilled water or nutrient solution. Kamat (327) found that 56 per cent of the sporidia germinated by a promycelium in distilled water and only two per cent by direct germ tube. On the other hand he found that in nutrient media (one per cent potato dextrose) fourteen per cent germinated by promycelium and eighty-two per cent by direct tube. Under favourable conditions he found that the aerial hyphae produced abundant conidia in chains or clusters.

Ajrekar and Likhite (13) state that infection takes place in the flowering stage but they found no dormant mycelium. It has been stated that it is the spore balls that furnish the source of material for infection in the case of bajra and that the flowers are the only parts infected.

Control. Ajrekar and Likhite (13) did not find seed treatment effective against long smut. Fortunately the smut is of little importance so that there is little need for specific control measures.

Sorghum Rust

Host plants. The sorghum rust is found on jowar, as well as other species of sorghum, on Sudan grass, Johnson grass and species of the genus *Holcus*.

Geographical distribution. It appears to be worldwide wherever *Sorghum* varieties are grown.

Appearance on the host plant. The rust differs from the other rusts in colour, being bright purplish red. The pustules are elongated parellel to the veins of the leaves.

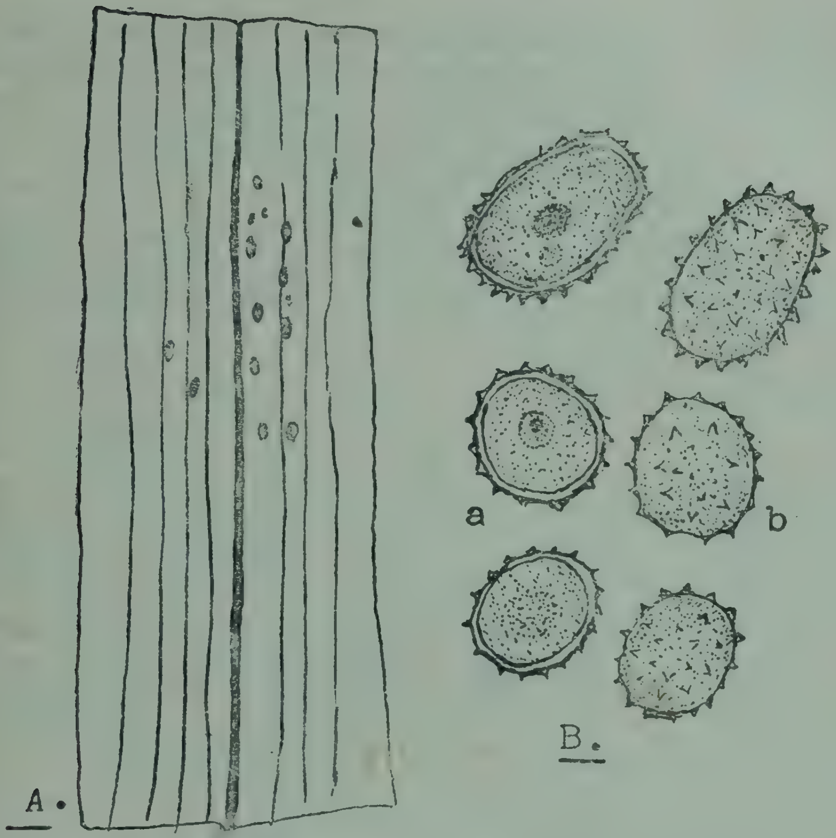


Diagram illustrating *Puccinia purpurea* on jowar leaf.
A. Diagram of infected leaf.
B. a. uredospores showing contents. b. uredospores showing spines.

The organism. Puccinia purpurea Cke.

Uredosori are scattered in irregular groups and are covered with the epidermis until mature. The uredospores, which are borne on long pedicels, are oval to elliptical with the base somewhat flattened where they are attached to the pedicel, and measure from 5 to 10 by 20 to 30 microns. They have fine spines on the walls and possess two circles of germpores about the middle. There are many large, club-shaped paraphyses which vary in colour from pale yellow to brown.

Teleutosori are found later in the same purplish spots as the uredosori. They are larger and when separate remain covered for some time. Teleutospores are dark brown ellipsoid to oblong with both ends rounded and with a slight constriction at the septum.

The Zonate Leaf Spot of Sorghum

Hosts. Sorghum vulgare Pers.

Geographic distribution. So far it appears to have been reported in only two places. Bain reported it in

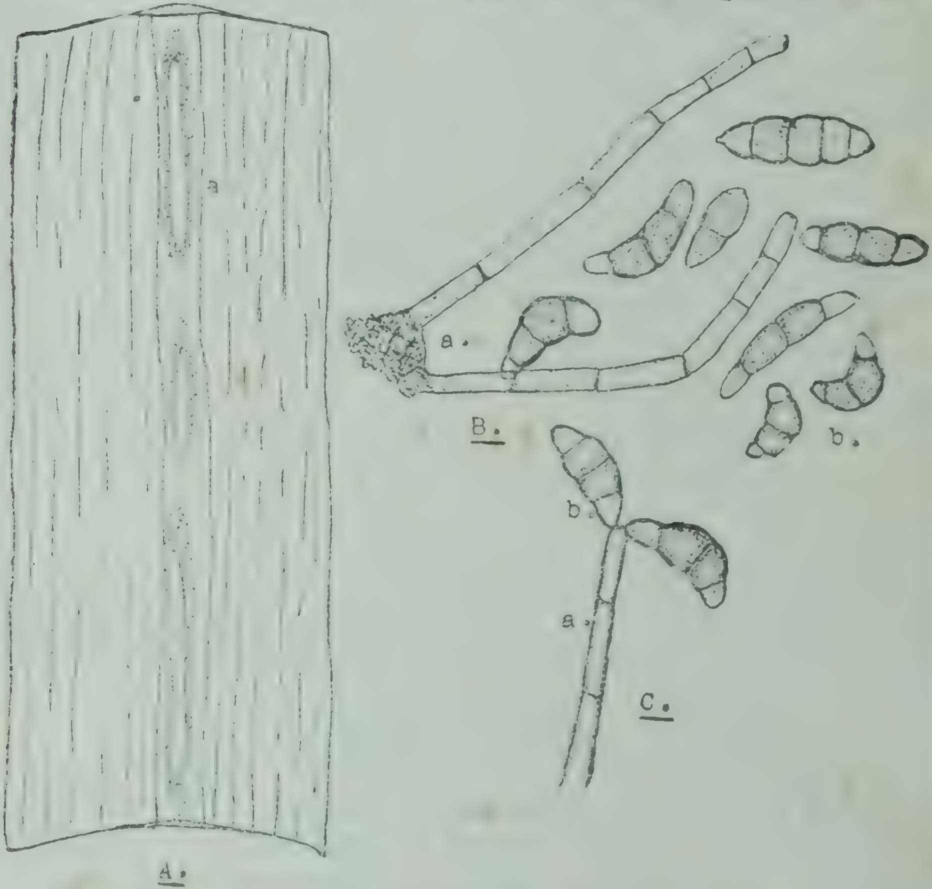


Diagram illustrating *Curvularia sp.* on sugarcane, jowar and maize. The species isolated from the three farm crops appeared identical. Note that the spots are similar to those of red spot of Sorghum but differ in that there are no setae in the case of Curvularia.

A. Diagram illustrating the infection area a.
B. Conidiophores. b. conidia.
C. a. Single conidiophore. b. Attached conidium.

the U. S. (39) and Mehta and Bose in India (459). It appears to be more or less general in certain areas in the two countries.

Appearance on the host plant. At first the spots are yellowish in colour, later becoming buff and then gray. The margins become dark and there may be a slight zonation. The spots may have a sooty appearance and this led Bain (39) to name it the sooty stripe disease.

The organism. The fungus is known as *Titaeospora andropogonis.* (Miuri) Tai.

The spots vary in size but the average is about 2½ inches long by ½ inch wide. Conidiophores are formed from the sub-epidermal stroma. They do emerge from the tissue of the leaf but produce the conidia when level with the epidermis. Conidiophores are hyaline and non septate. Mehta and Bose (459) have given the measurements as 22—35 × 3—3.5 microns for those found at Cawnpore.

Conidia are borne at the tips of the conidiophores. They are slender, cylindric, 1-8 septate and may be branched. Measurements vary with the conditions of growth, varying from 50 to 110 microns. The average at Allahabad appeared to lie between 65 and 75 microns.

Following conidial formation, dark sclerotial masses are formed. Mehta and Bose (459) observe that conidial formation is scarce and it would appear that it is of short duration. The fungus grows readily on ordinary media but the conidial production was not observed.

Isolations made at the Agricultural Institute, Allahabad, have been sent to the Type Culture Collection, New Delhi.

Control. At this time there have been no control measures worked out. The fungus appears to be seasonal and it is hoped that present observations on weather and fungi will produce some valuable information about the requirements and thus offer a suggestion as to possible control.

Helminthosporium Leaf Spot of Jowar

Hosts. Maize, jowar, wheat, barley, oats and sugar-cane.

Geographic distribution. Apparently world-wide.

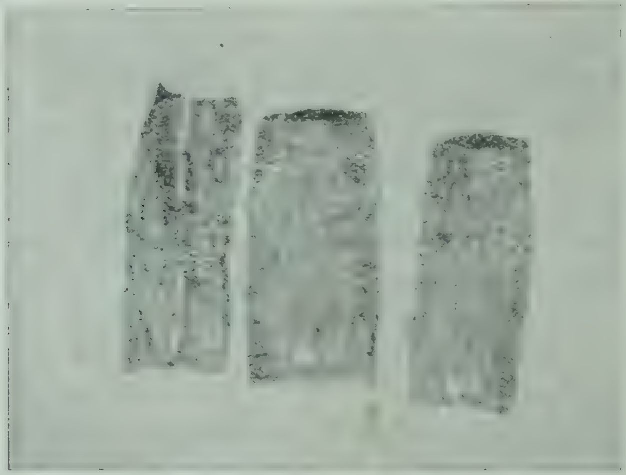


Photo showing the typical infection spots of *Helminthosporium turcicum* on leaves of Sorghum.

Appearance on the host plant. The general symptoms are the same as for that on maize except that the spots are somewhat smaller. As the spots tend to extend along the leaf and to lie between the main veins it may be that vein limitation of the spots would make them somewhat more narrow than on maize.

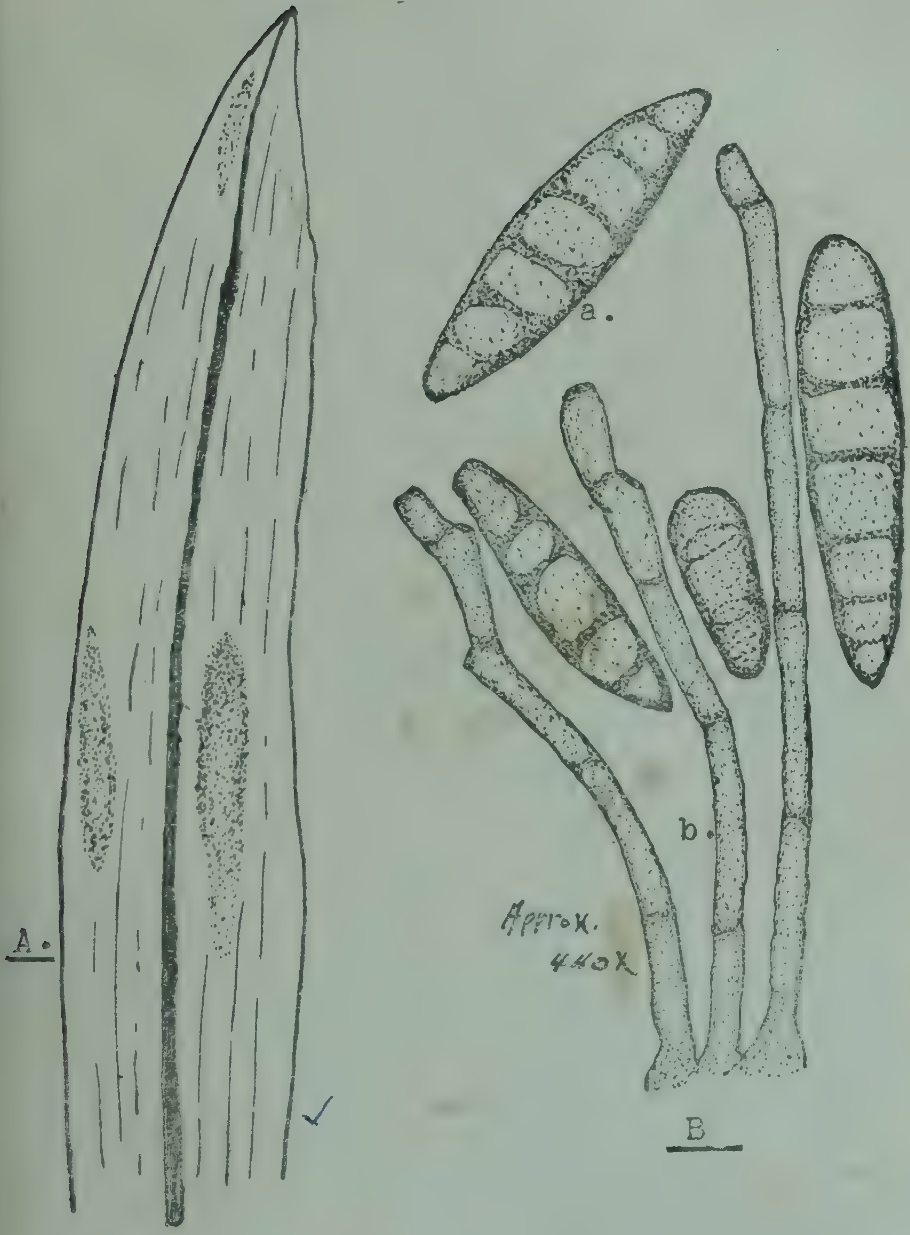
The organism. *Helminthosporium turcicum* Pass.

For description of the fungus see under maize.

Control. Sanitation and rotation are the only suggested control measures at this time.

Root Rot of Sorghum

Hosts. Very wide range.



Approx.

Diagram of jowar leaf illustrating infection by *Helminthosporium* leaf spot.

A. Diagram of an infected leaf.

B. a. Conidia. b. Conidiophores.

Geographic distribution. World-wide.

Appearance on the host plant. The characters are that of poor colour, reduced size and weak stem. When root rotting is bad the plants may lodge easily or be easily pulled from the ground. Examination of the roots will show that most of them are gone and those that do remain are badly discoloured and often more or less completely disintegrated. If one examines a plant during the first stages of the attack it will be noticed that there are many discoloured spots on the roots. From these may be isolated a number of fungi which may, either alone or together, cause the typical root rot symptoms. Root rotting is much worse in low ground where drainage is poor. Jowar does not like to have "wet feet" as the saying goes.

The organism. First on the list is *Macrophomina phaseoli* (Maubl.) Ashby. The stage most often found, however, will be *Sclerotium bataticola* Taub. Others likely to be found will be *Rhizoctonia solani* Kuhn., species of *Fusarium*, *Pythium* species and occasionally *Sclerotium rolfsii* Sacc.

Control. Well-drained fields. Possible rotation or fallow. Probably organic manure will help by building up the soil flora to combat the root rotting organisms.

Note: Uppal (842) reports a stem disease of jowar which he says is due to *Macrophomina phaseoli* that caused considerable loss in the Gujerat area. Early maturity were the main symptoms. When the stems were split open the sclerotia of the fungus were evident on the pith. An epiphytotic developed among the seedlings and some loss occurred. The fungus gained entrance to the plant by way of the roots and sometimes passed up to the top. The only outward symptom was the early maturity, already referred to, and the peculiar hollow sound of the stems when struck or blown by the wind.

Sclerotia measured 110-130 microns which placed the strain in Haigh's group (see under Cotton Root Rot)

C. The pycnospores measured 10—24×6—10 microns. The optimum temperature was 35°C.

THE COMMON DISEASES OF BAJRA

Bajra, like jowar, is subject to a number of diseases, the most serious of which would probably be downy mildew, smut, rust and root rotting. These will be discussed separately.

Downy Mildew of Bajra

Hosts. The mildew has been found on many of the millets and closely related species of grasses. So far as is known, no species or variety of maize is immune. Melhus and van Haltern (470) inoculated dent, pop, sugar and flint corn successfully with the oospores. Mitra (479) reported the disease on sugarcane Co. 316 in 1931. In nature it appears to be able to live on species of Setaria and thus there is always a source of inoculum for the cultivated crops. In India it appears to be most serious on bajra (*Pennisetum typhoideum*).

Geographical distribution. It is probably worldwide. That it was considered to be serious is evidenced by the quarantine acts which prevented the importation of any portion of Indian corn (maize) into the United States from south-eastern Asia (including India, Siam, Indo-China and China), the Malaya archipelago, Australia, New Zealand, the Philippine Islands, Formosa, Japan and adjacent islands (96). These acts applied as well to the closely related species of teosinte and covered importation of plants or plant parts in the raw or manufactured state, on account of downy mildew. This was in 1921. In 1929 Rhodesia passed a quarantine act prohibiting the importation of maize seed from India, the Philippine Islands, the Dutch East Indies and other countries affected with *Sclerospora.* Porter (616) reported it as widespread in China, especially in north China. It has been reported from many parts of India and on many of the grasses.

Appearance on the host plant. Inoculation experiments show that infection may take place in the early seedling stage and that it may be at this time most of the infections do take place. Frequently the first leaf will not be infected but the second, third and all others may.

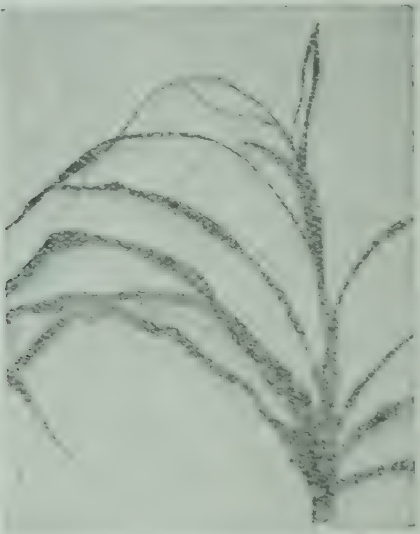


Photo of a diseased bajra plant showing typical shredding of the leaves as a result of the fungus action.

The first appearance of disease will be a slightly lighter colour which will be found in streaks parallel to the midrib and usually about half-way between the midrib and the leaf margin. As the disease progresses the colour becomes yellow and then brown. The streaks dry and then break so that they shred and become whipped into long brown fibres, the vascular bundles being the tissues which remain intact and the softer parenchyma tissues rupturing.

If the terminal bud is killed the other buds may grow and the plant may have several branches. This may go on in some cases until a condition resembling witch's broom results. In bajra this is quite often the case. When the plant heads, if it does, the spike may be distorted and the spikelets be elongated so that the whole floral structure gives the appearance of an ear with long hairs over it. The colour of the spike is at first green and this peculiar structure is known as "green ear". Later it turns brown and dries.

The organism. The fungus is known as *Sclerospora gramnicola* (Sacc.). Schroter. There appears to be biologic specialization in the fungus. The one on bajra will not attack jowar and *vice versa.* Weston (928) found

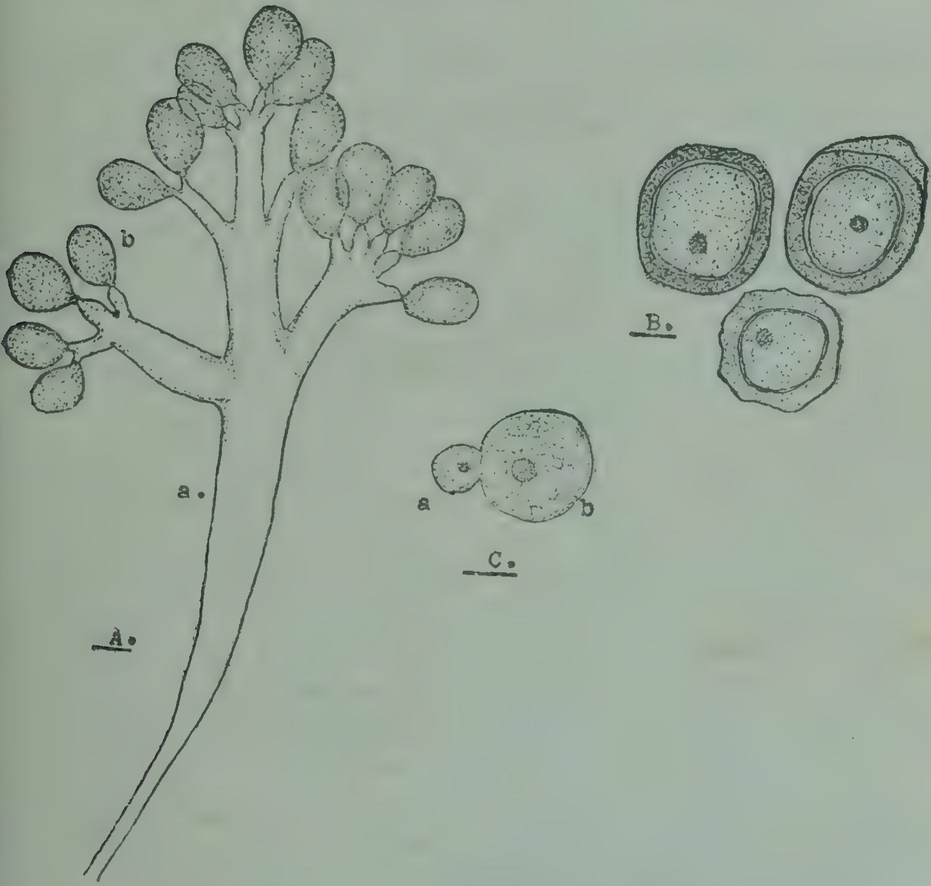


Diagram illustrating downy mildew of bajra and maize, *Sclerospora gramnicola.* (Drawn by K. B. Pisharodi).

A. a. Conidiophore. b. Conidia.
B. Oospores.
C. a. Antheridium. b. Oogonium.

that the one on sorghum differed from that on Setaria and that the difference was sufficient to consider the species distinct and he accordingly raised it to specific rank, calling it *Sclerospora sorghi.* Uppal and Weston (929) after a study of the philippine form in comparison with the Indian form known as *Sclerospora indica* concluded that there is no basis for the two species and therefore merged them both into S. *philippinensis* Weston.

The conidiophores are produced on the surface of the host plant some few hours after the first symptoms appear. They are about 100 microns in length, sometimes more, with an average diameter of 12—15 microns. They are branched at the top into short, rapidly tapering branches which bear conidia at the tip. The conidia, often referred to as sporangia, are hyaline, broadly elliptical, slightly pointed at the free end, with a thin wall and measure 19—31 × 12—21 microns. They are only lightly attached to the conidiophore and fall early. They germinate rapidly in water, producing 3—12 zoospores. They lose their vitality soon if dry.

Later in the same areas the sexual stages are formed. The female reproductive body is the typical oogonium, thin-walled, hyaline at first, soon becoming lightly brown. Each contains a large nucleus and dense cytoplasm. The male reproductive organ is a much smaller structure and may be identified by the fact that it is either twined around the oogonium stalk or in contact with the wall of oogonium itself. Fertilization takes place by a breaking down of the wall and the formation of a conjugation tube through which the male gamete passes into the oogonium. After fertilization the oospore becomes much more dense and the wall of the oogonium thickens and becomes darker in colour. Often an amber brown colour is seen by trasmitted light. The oospores are spherical with a uniformly smooth wall. The oogonium wall on the outside, however, may be very rough so that the appearance of the whole body may be irregular and rough. The oospores are some 22.5—35 microns in diameter. Germination is by germ tubes. Various methods for germination of the oospores have been suggested. One of the simplest is that of Evans and Harrar (212) which requires only that the oospores be placed in sterile distilled water at room temperature, the spores germinating in about 24 hours.

Life cycle. From all data so far gathered it appears that the fungus lives over in the oospore stage and that it is the agent of infection as the new crop appears. Anstead (29), Uppal (840) and Uppal and Weston (928) have shown that the oospores are the agents of infection in the early spring. It is not considered that the mycelium can live over in the seed.

As the young seedlings emerge in the early season the oospores germinate and, if in contact with the seedling, infection takes place at once. The progress of the disease and the symptoms are noted above.

Conidia are produced in large numbers and are spread over the same and surrounding plants by wind and water. It has been held that most of the conidia are produced at night and that they do not retain their vitality for long. Under favourable conditions the disease will spread rapidly from plant to plant.

As the diseased areas age and become brown the oospores are produced and as resting spores they may live for months in the old leaf and stalk material or in the soil, until conditions for germination are restored, and then germinate to infect the susceptible host that may be present.

Control. About the only control which appears effective at present is clean cultivation and eradication of all host plants from the neighbourhood, coupled with rotation of crops. Weston (928) suggested a seed treatment which he said was successful. The seeds were first wet with alcohol for half a minute and then covered with concentrated H_2SO_4 for ten minutes, after which they were washed free of acid in running water, dried and planted. He reported that germination was only slightly reduced by the treatment but that the downy mildew was greatly reduced.

The Rust of Bajra

Host plants. This rust occurs on the millets of which bajra is the most important host.

Geographical distribution. The rust has been reported in Africa and in the Madras, Bombay and Central Provinces regions of India.

Appearance on the host plant. The sori are brownish yellow in colour and often arranged in linear rows on leaf and stem. On the young leaves there may be a brown area about the sori but this does not appear on the older leaves. The teleutosori are darker than the uredosori.

The organism. Puccinia penniseti Zimm.

Uredosori appear on both surfaces of the leaves and may run together so that they appear like flakes. The uredospores are oval, the walls are covered with scattered spines, possess four equatorial germ pores and are supported on colourless pedicels. They measure 33—38 by 23—30 microns.

Telia are similar to the uredinia except that they are brown. The teleutospores are cylindrical, more or less flattened at the tip, possess a smooth thickened wall, sometimes constricted at the septum and measuring 40—60 by 16—20 microns. No paraphyses are known among these spore forms.

The aecial stage is unknown and it is probable that the fungus does not need it to carry out its life cycle.

Life cycle. Only uredo and teleuto stages are known which would indicate that the fungus lives over the dormant period in the uredospore stage. Destruction of all diseased leaves and leaf sheaths will be helpful in control where the disease is serious. It usually does not appear until late in the season and thus it does little damage.

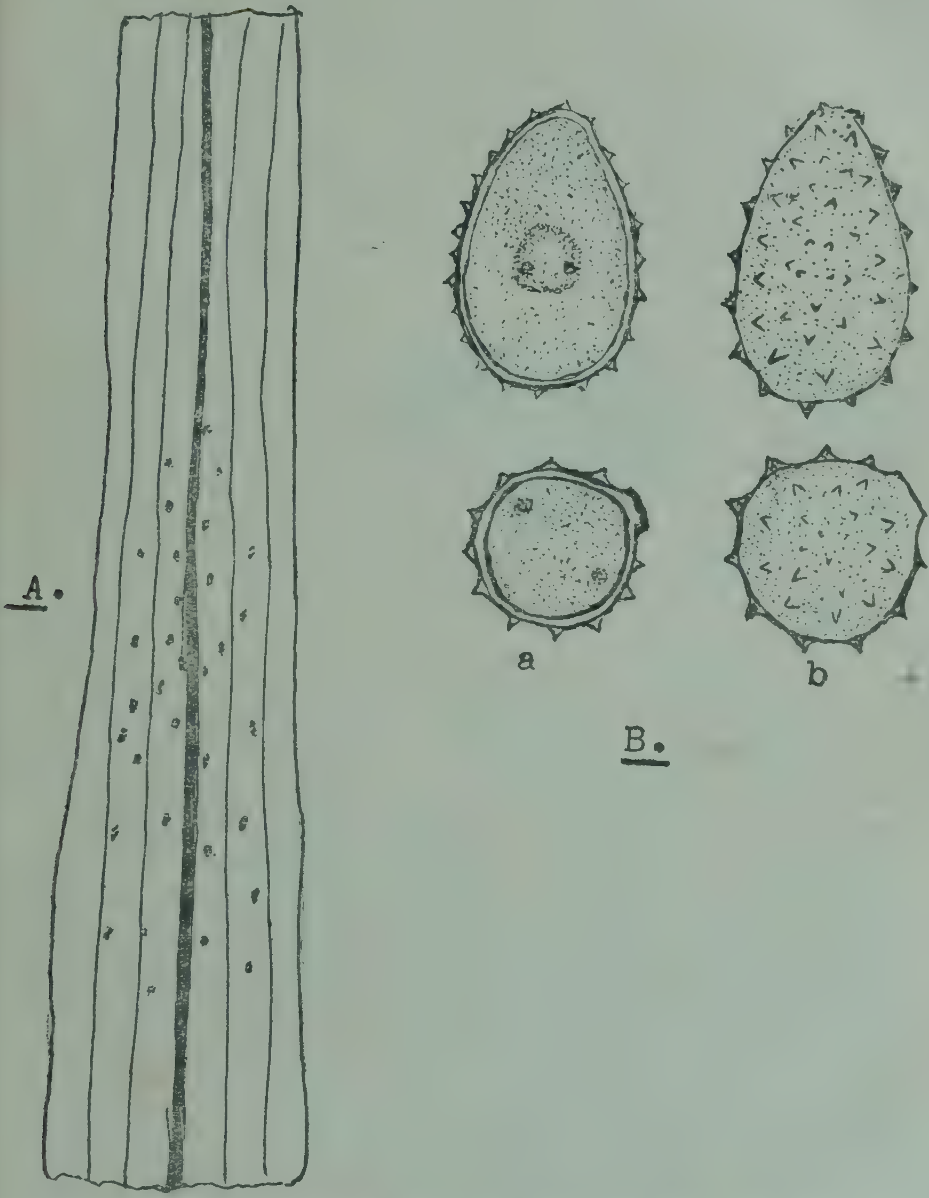


Diagram illustrating the rust, *Puccinia penniseti* on a bajra leaf.

A. Diagram of infected leaf.
B. a. Uredospores showing contents. b. Uredospores showing spines.

Control. Usually not severe and special control measures not needed. Destroy old infected leaves and stalks. Rotation.

Common smut of Bajra

Host. It occurs on millets.

Geographic distribution. General over Africa and Asia. In India it is common in most areas where bajra is grown but is most severe in the Madras and Sind areas.

Appearance on the host plant. On the bajra spike the first appearance of the smut is enlarged kernels which are green in colour. These are two to three times the size of the normal kernels. As time goes on the green kernels become black and when crushed are found to be filled with a mass of black spores. In the field it is easy to detect the smutted spikes by the size of the kernels.

The organism. Tolyposporium penicillariae Bref.

The spores are held together in masses or balls by a mucilaginous material and by a surrounding membrane. Ajrekar and Likhite (13) state that infection takes place during the flowering stage but they found no dormant mycelium. McRae stated that the spore balls were the source of infection for bajra and that infection took place through the flowers. Seed treatment according to Ajrekar and Likhite (13) did not control the smut but the disease is of little importance except in local areas.

Control. Rotation and destruction of the smutted spikes.

Root Rotting Fungi on Bajra

The roots of bajra like those of jowar are also destroyed by root rotting fungi and probably in most cases by the same fungi. Under some circumstances heavy loses of fodder may occur due to the root rotting fungi which may become stem rotting as well. Species of *Fusarium*, *Rhizoctonia*, *Pythium*, *Sclerotium bataticola*, as well as many fungi thought to be only saprophytes, have been isolated from the diseased bajra roots. In one case of stem rotting, *Fusarium poae* was isolated. This was identified by Dr. G. Watts Padwick, then Imperial Mycologist, Imperial Agricultural Research Institute, Delhi.

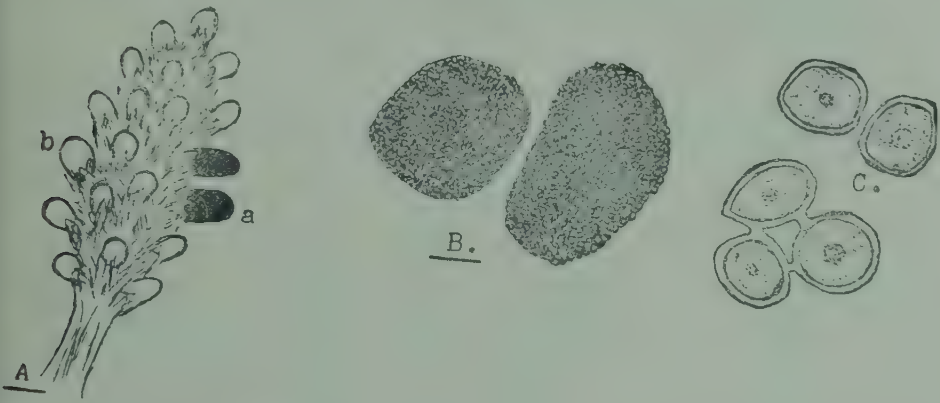


Diagram illustrating the grain smut of bajra.

A. Portion of a panicle with. a. Smutted grain. b. normal grain.
B. Mass of smut spores.
C. Chlamydospores enlarged.

The problem of control is the main worry of the farmer. As in the case of the root-rotting fungi of jowar and other cereal crops, good drainage is one of the most essential things. It is probably best not to follow bajra with bajra in the rotation as this tends to build up the particular fungi that attack the roots. Little disease resistance among the bajra varieties is known so that for the present control rests mostly upon cultural methods.

THE COMMON DISEASES OF MAIZE

Maize is subject to a number of diseases. Among the most common in India are rust, common smut

(*Ustilago zeae*) *Helminthosporium* leaf blight, *Curvularia* leaf spot, (See under sugarcane) head smut (*Sorosporium reilianum*), and root rotting.

Leaf Blight of Maize

Host plants. Host plants are maize, Jowar, wheat, barley and oats while sugarcane Mitra (483) may also be infected.

Geographic distribution. It appears to be worldwide wherever maize is grown.

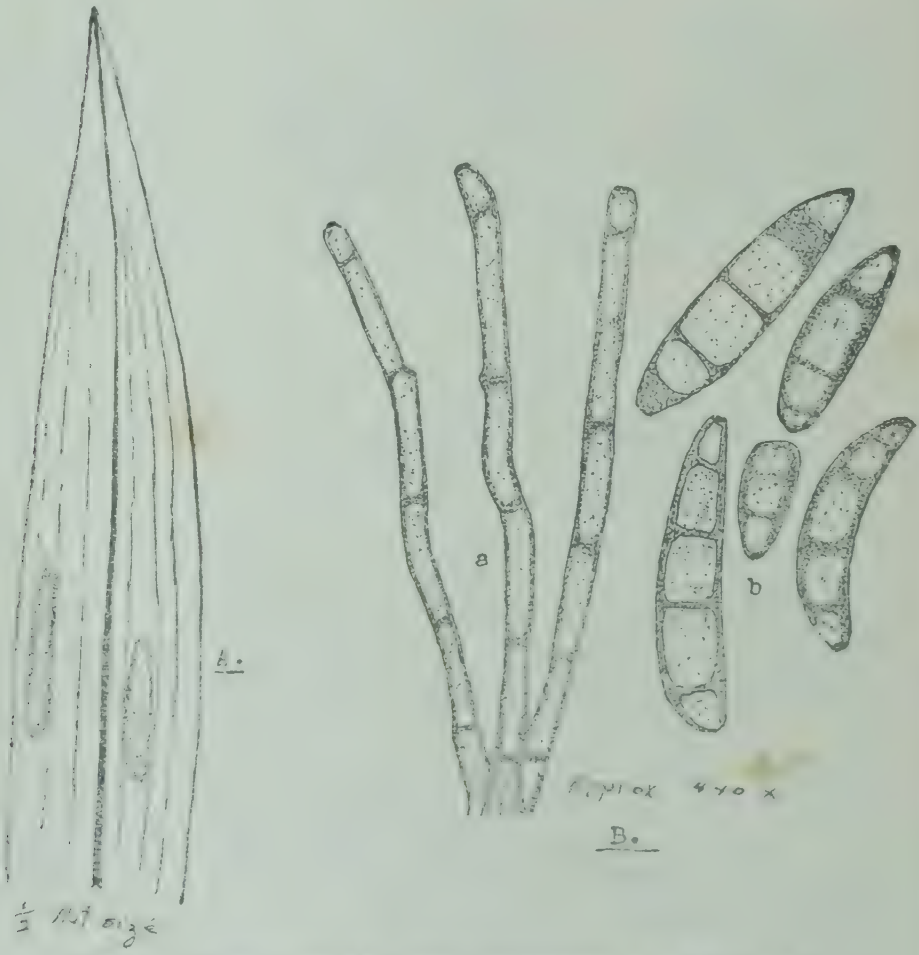


Diagram illustrating the Helminthosporium leaf spot of maize.
A. Diagram of an infected leaf.
B. a. Conidiophores. b. Conidia.

Appearance on the host plant. The first appearance of the fungus is small, yellowish, round or oval spots on the leaves. These may extend along the leaf for some distance, being limited by the larger veins. The spots may coalesce into bands. In moist weather the spots become darker and larger numbers of conidiophores and conidia, which are dark greenish in colour, form over them.

If the attack is severe, the plants may be stunted and ears poorly developed. The straw coloured spots may include the whole leaf. In some cases the ears rot with a loose matted black mycelium present.

The organism. *Helminthosporium turicicum* Pass. It has also been called *H. inconspicuum* C. & E.

The conidiophores appear in groups of 4 to 6 through the stomata. They are long 24 to 260 microns and average 7 to 9 microns wide, septate and usually unbranched and yellowish brown in colour. They measure 45 to 132 by 15 to 25 microns and possess from 1 to 8 septa.

The seasonal development of the fungus is similar to the species of *Helminthosporium* referred to on barley and wheat. Butler (93) states that it was not serious on maize then and it does not appear to be so at this time.

Control. So far the only control is sanitation and rotation.

There are a number of other sepecies of *Helminthosporium* on various crops in India at this time that are not of much importance. *Helminthosporium sacchari* Butl. has been reported on sugarcane. Mitra (483) believes that the strains on jowar and maize are biologic forms.

Smut of Maize

Host plants. The maize smut is found on the species and varieties of *Zea*.

Geographic distribution. The smut is world-wide but appears to be worse in some sections than in others. In India the most serious damage is done in Kashmir and the colder sections where maize is grown.

Appearance on the host plant. The extensive hypertrophy caused in the infected parts of the host plant make it easy to identify. All parts of the plant are subject to infection and galls may appear on any of the above ground portions. The sori are more conspicuous on the ears and staminate inflorescence than on other parts of the plant but they also occur on leaves, sheaths and stems.

The organism. Ustilago zeae (Beckm.) Unger.

It has been called by other names. Beckman called it a *Lycoperdon*, apparently because of the resemblance of the sori to puff-balls. It has also been referred to as *U. zeae maydis* but the present accepted name is the one given above.

Ustilago zeae differs from most of the smuts in the manner of infection. It is not a systemic disease but is confined to local areas in the neighbourhood of the infections.

The tissue of a smut gall consists of short cells which are inclosed in a gelatinous matrix when young but become free upon maturity. The mature chlamydospores are nearly round brownish and possess spiny walls. They measure from 7 to 12 microns in diameter.

Upon germination the spores produce promycelia which vary in the number of cells possessed. Recent investigations indicate that the number is not 4, as is typical of the species of *Ustilago*, but that it may be anywhere from 1 to 4 with 3 the more common number. Under favourable conditions the promycelium may branch and produce a number of primary sporidia which may in turn bud to produce a number of secondary and tertiary sporidia, all of which are capable of infecting the maize plant.

Infections by the sporidia take place through the epidermis of leaf and stem or by way of the pistillate and staminate flowers. The surface of the plant is protected by a cuticle which has a high surface tension and this prevents many of the sporidia from infecting the plant. It has been found that if this is reduced by any substance like soap, resin or other agent, the number of infections may be greatly increased.

The sporidia are haploid when discharged from the promycelium and infection hyphae are also haploid. This haploid mycelium is capable of a weak infection but cannot produce galls. The diploid mycelium is capable of producing sori on the maize plant. It was the discovery of this fact by Hanna (259) which led to our present knowledge of the heterothallic condition of *U. zeae*. He found that if he sowed monosporidial (haploid) cultures of smut on maize that infections took place but no galls were formed. On the other hand if he sowed mixed cultures of the smut sporidia on the maize plants galls formed. Thus there must be sporidia of both sex groups present before true sori (galls) are formed.

Hanna (259) also was able to demonstrate that there are two kinds of sporidia formed on the promycelium representing the two sex groups. Just before germination of the chlamydospore the two nuclei present in the spore fuse and then there is a meiotic division to form the haploid nuclei and this is followed by a mitotic division to form the sporidia. The sporidia are formed in equal number so that in the distribution of these gamete cells they are scattered over the maize plants at random. It is evident that in nature many of the sporidia fall on a leaf alone and thus never produce sori. But as there are many of them some would by chance fall onto a leaf in the immediate neighbourhood of sporidia of the apposite sex group and thus mycelium fusion would take place and galls form. From what has been said it is easy to work out the life cycle of the smut.

Stakman (at Univ. of Minn. U. S. A.) has done an immense amount of research on *U. zeae* and according to his work *U. zeae* is made up of a number of lines or strains which are capable of segregation and usable for breeding purposes. He has found mutable and constant lines which, when crossed, would produce progeny in three groups conforming to simple Mendelian ratios.

Control. Sanitation is of importance. The destruction of all the diseased parts, either by ploughing under or burning, removes one of the chief sources of infection.

Smut resistant varieties have been developed in the United States and are probably the chief hope of the farmer. In India little breeding work has been done so far.

Johnson (316) found that certain bacteria are antagonistic to smut and this may offer a possible control.

Note: Wittich and Stakman (935) report an interesting case of a 48-year old man who had suffered with asthma for 20 years and when examined the spores of *U. zeae* were found in his sputum. When tested he gave a positive reaction to *Tilletiai tritici, T. foetans, Ustilago tritici, U. hordei, U. nuda, U. avenae, U. crameri, U. zeae, Urocystis occulta, Sphacelotheca sorghi, S. cruenta* as well as a number of rusts. This is of interest as it may offer an explanation for a number of the bronchial troubles that affect men working with various plant products.

Head Smut of Maize

Host plants. Maize and sorghum varieties are the principal hosts of this fungus.

Geographical distribution. It is more or less common in parts of Russia, Australia and India. Occasionally reported in the United States.

Appearance on the Host Plant. As indicated by the name, this smut is found in the pistillate and the staminate inflorescences, Tumours are formed in place of the flowers and this is one distinction between this smut. and *Ustilago zeae.* The floral parts become a powdery mass of spores.

The organism. Sorosporium reilianum (Kuhn) McAlpine. It has also been known as *Ustilago reilianum* Kuhn and *Sphacelotheca reilianum* (Kuhn) Clinton.

The first appearance of the smut is a mass of hard, rough tissue in the inflorescence which is in striking contrast to the soft tissues of the maize smut. Where sori form the parts become enlarged and deformed. According to Butler (93), where the staminate inflorescence is infected the ears are likely to be infected also. Occasionally broom like structures may form in the place of the normal inflorescence.

The sori vary in size, occasionally reaching several inches in length. As in the case of *Ustilago zeae,* the spore mass replaces the host tissues. The spores are reddish brown to black with finely echinulate walls and measure from 10 to 16 microns in diameter. Mixed with the chlamydospores are numerous sterile colourless cells. The spores germinate to produce a typical promycelium and sporidia. Secondary branching of the promycelium as well as budding of the sporidia may occur under favourable circumstances.

Infection may occur from seed-borne spores but the principal source of infection is the soil-borne spores. Sporidia and promycelia are produced at the time of germination of the seed and infection usually takes place while the tiny plant is emerging from the seed. It may, however, take place after the plant has unfolded its first leaves. Floral infection may occur in the terminal as well as the lateral buds.

Control. Sanitation and rotation are the most important methods of control since the spores are not generally seed borne. In the United States resistant varieties have been developed which offer hope of control. In India little has been done along this line as yet.

Maize Rust

Host plants. Maize and related fodder plants which include teosinte (*Euchlaena mexicana*).

Geographical distribution. The rust has been found in the United States, Europe, Africa, Australia and India. It has been reported as most common in the Bombay region but occasionally in other districts. Bryce reported as present in Ceylon.

Appearance on the host plant. The uredosori appear on both surfaces of the leaves, either scattered or in groups. When severe, the leaves lose their colour, dry and wilt to such an extent that the ears may not develop.

The organism. Puccina maydis Bereng. *Puccinia sorghi* Schw.

The uredosori, which are principally on the leaves, are first small, yellow spots which later elongate and become reddish brown, finally becoming pustular. These rupture and the reddish brown spores are exposed. The uredospores are ovate or globose, finely echinulate and 23—30 by 22—26 microns. The teleutosori are dark brown but other wise like the uredosori. The teleutospores are ovate oblong, constricted at the septa, chestnut brown in colour and 28—45 by 12—17 microns is size. They possess a persistent pedicel which is from once to twice the length of the spore.

The aecidial stage is found on three species of *Oxalis*, namely; *Oxalis corniculata*, *O. europea* and *O. cymosa*. The aecia are citron yellow in colour, sometimes orange with a raised margin of leaf tissue as a border. It is thought that the aecial hosts may play a definite roll in the cold sections as the uredospores have not been found surviving the winters.

Physiologic races exist and Dickson (190) page 55, presents a key which includes the 7 known races.

Life cycle. The complete life cycle would be; sporidia to *Oxalis;* the production of pycnia and oogonia followed by the formation of aecia. Aeciospores to maize and the production of uredospores and teleutospores on maize.

Control. So far no real control has been devised. One might expect to look for resistant varieties since that is the promising source of control.

Seed, Seedling and Root Rot Diseases of Maize and Sorghum

Although the three groups of diseases referred to in the heading are more or less distinct yet they are sufficiently alike to warrant their being discussed under the same general heading. Seed decay fungi may be limited to the seed but they may also invade the seedlings and even attack the root system later. Many of the fungi will be found on the seeds and seedlings of many of the cereals. Fungi not present on the seeds but present in the soil may attack the seedlings and *vice versa.* Many of those on the seedlings that do not kill the seedlings may persist onto the roots of the mature plants. There are likely to be some fungi present in the soil that will attack the root systems after the plants have become several inches high which did not attack the seedlings.

Seeds surface sterlized and plated out on agar or sterile blotters will develop moulds of a wide range of species. In this area they are likely to be species of *Mucor, Aspergillus niger, Penicillium* species, *Curvularia lunata, Altenaria* species, *Helminthosporium* species, as well as numerous other saprophytic forms. These are common to nearly all of the cereals but the species will differ with the different species of grain. The portion of the seed most commonly infected by the parasitic

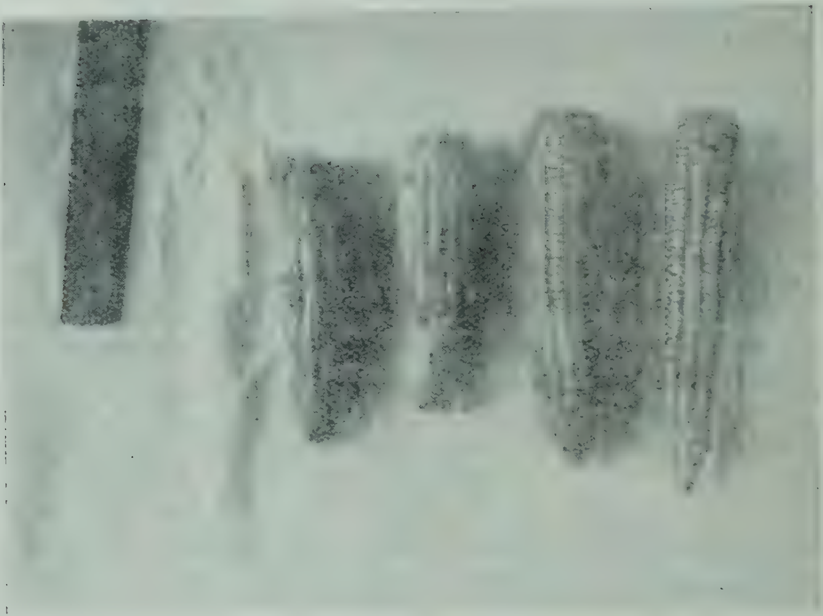


Photo illustrating diseased and healthy ears of maize. From left to right; ear damaged by Fusarium species in the field; ear damaged from heating in storage and two normal ears.

Photo of plate planted with seed from ear on extreme left.

Photo of plate planted with seed from normal ear on right.

species is the scutellum, the single cotyledon of the monocotyledon seed.

Kohler and Holbert (349) have given a number of fungi as having been found on the scutellum and other portions of the seed. Among the common fungi they found; *Rhizopus* spp. *Fusarium moniliforme*, *Cladosporium acremonium*, *Aspergillus* spp., *Penicillium* spp., *Alernaria* spp. and others. All of them have been observed on the germinating seeds of maize and jowar at Allahabad as well as some which they did not observe, especially *Curvularia lunata*, which is common here. Many of these same fungi are found on seedlings. On very young seedlings the mesocotyl, that portion between the first and second nodes of the seedling or that portion between the main root system and the adventitious roots, is likely to be attacked. *Aspergillus niger*, *Sclerospora gramnicola*, *Pythium* species, and *Penicillium species* are likely to be found. Injured seedlings are likely to show a pale green colour and be dwarfed and distorted *Aspergillus* infections, according to Kohler and Holbert, are likely to be a lighter green in colour whereas those infected by *Penicillium* are more likely to be normal in colour but with distorted stalk and leaves. *Sclerospora* infections do not appear to come from the seed but from the soil. Infection of seedlings is not common and when it occurs the seedlings dies young.

Root rot, of the maize, sorghum and sugarcane group, is caused by the same general group of fungi with some additions. The fungi most commonly isolated from the diseased roots of the three plants mentioned, have been species of *Rhizoctonia* and *Fusarium*. *Rhizoctonia solani* and *Macrophomina phaseoli* are common. *Sclerotium rolfsii* and *Pythium* species have also been found but indications are that they do not cause much damage after the seedling stage.

Symptoms caused by the specific root-rot organisms are hard to determine. About the only sure way is to isolate and identify the fungi in the laboratory. Plants suffering from root rot are likely to show stunting and wilting, especially in dry weather. Because of a damaged root system they show wilting first and are also likely to mature first in the field. Older leaves may die and turn brown prematurely. If the plants are removed from the soil they will pull up easily and on examination show practically no root system and those that are found will be short, show considerable blackened surface area and local diseased regions. The primary roots are likely to be completely destroyed with only secondary or even teritary roots remaining alive. In the state of Iowa, U. S. A., in 1944, there had been a very long rainy season during June and the early part of July Corn fields were flooded and many were under water for days so that the roots were completely covered. Many died but those that survived had no root system left. Most of the plants could be pulled easily from the soil. At the time of the ending of the heavy rains many of the fields had been given up as hopeless. However, from then on the rains came regularly and spaced so that the ground was kept damp but not flooded. The plants put out secondary roots and later the prop roots developed a dense fibrous root system. Many of the fields, which had been declared by their owners as lost in June and July, came through with fifty, sixty, and seventy bushels of corn an acre. Not a high yield but certainly better than expected and the yield was evidence of the power of recuperation of the corn plant. No general attempt was made to isolate the fungi concerned but from other sections of the corn area Pythium species, *Fusarium* species and *Rhizoctonia* were most common.

Reports of the various fungi on cereal roots have been made from time to time. Padwick (568) in 1940 reported a *Pythium* species on maize seedlings in Bihar. Uppal (849) reported *Macrophomina phaseoli* on sorghum in Bombay in 1935. He found that it reduced the heads to one-fifth their normal size. Subramaniam (758) found a *Pythium* on sugarcane seedlings which he decided was *P. gramnicolum.* He also found the same organism on maize and sorghum seedlings. He also reported *Helminthosporium halodes* on sugarcane seedlings. Likhite reported in 1936 that he had found *Macrophomina phaseoli* going from cotton seedlings to sorghum. Uppal et al (853) found that *M. phaseoli* was able to attack sorghum. They determined that infection took place on the feeding roots. The optimum temperature for the fungus was 35.5 °C. At that temperature seedlings infection was highest. High soil moisture was also found optimum for the fungus. Ramakrishnan (640) found *Fusarium moniliforme* causing twisted top of sorghum. Mitra recorded it on sorghum at Allahabad and it has been found on maize ears at Allahabad.

A summary of the fungi found on the seeds, seedlings and root systems of the three plants under discussion would be about as follows:

Alternaria species.
Aspergillus niger van Tiegh.
Curvularia lunata (*Walker*) Boedijn.
Fusarium moniliforme Shel.
Helminthosporium sp.
Macrophomina phaseoli (Maubl.) Ashby.
Rhizoctonia solani Kuhn.
Rhizopus species.
Pythium species.
Pythium gramnicolum Subramaniam.
Sclerotium rolfsii Sacc.

Control. Seed treatment has helped in the control of the seedling diseases. McLaughlin (435) reported in 1944 they had highly significant results on one-third of the farms in the U. S. A. on which they planted seed treated with Semesan Jr. at the rate of 3 oz. a bushel. Trials at the Institute in which Arasan, Barbac

C and Spergon were used on maize seed at the same rate gave increased yields in each case with Barbac C giving highest yields but no one of the three gave significantly higher yields than check. Perhaps organic manures and deep ploughing to place the decomposing material some four to six inches beneath the surface of the soil would be the best control. Resistant varieties are possible but because of the complexity of the soil flora the problem is an exceedingly tedious one and if it is possible to secure the organic matter it is a much more practical way of solving the problem.

A List of the Smuts of the Common Crop Plants

Dr. B. B. Mundkur has compiled the following list of smuts which are common on the major crop plants and it is included here with his kind permission. It will serve several purposes for the student. It is an accurate list with correct names and the manner of transmission. These are all found on most of the farms of the United Provinces and therefore among the fungi which the student of agriculture should know.

STUDIES IN INDIAN CEREAL SMUTS

VIII. NOMENCLATURE OF INDIAN SMUT FUNGI AND PROBABLE MODES OF THEIR TRANSMISSION

By B. B. MUNDKUR, Imperial Agricultural Research Institute, New Delhi

(*Received for publication on* 21 *December* 1944)

In a recent publication Stevenson and Johnson (1944) have reviewed the position regarding the names that should be applied to cereal smut fungi in order to strictly adhere to the International Rules of Nomenclature.

Previous contributions in this series appeared in *Proc. Indian Acad. Sci.* 11, 267-70 (1939); *Indian J. agric. Sci.* 11, 675-702 (1941); 13, 54-58, 631-633 (1943).

A majority of the Mycologists will be in complete accord with the conclusion of Stevenson and Johnson (1944). Their list of names deals with only those smuts that occur in the U. S. A. A list which includes the smuts affecting the Indian crop plants has therefore been compiled and presented below.

The list also includes the available information regarding the modes of transmission of the smut diseases, wherever they are known. In the study of plant diseases, a knowledge of their mode of transmission is very important, for without such knowledge, it is not possible to devise proper control measures. Smuts may be externally seed-borne or internally seed-borne, soil-borne or air-borne. Methods developed for controlling externally seed-borne smuts are worthless for controlling internally seed-borne smuts. For smuts that are air-borne seed treatments are of no value.

Unfortunately we do not yet know the mode of transmission of all the smuts affecting our crop plants. Recently the author (1943) has shown that the Karnal bunt is air-borne. Probably the bunt of rice is also air-borne. It is hoped that the Mycologists in whose areas such smuts occur will be stimulated to do further research so as to fill the lacunae in our knowledge of the transmission of such smuts.

Crop	Common name of smut	Scientific name according to		Mode of transmission
		International Rules of Botanical Nomenclature	Previous Practice	
Wheat (*Triticum* spp.)	Loose smut . .	*Ustilago tritici* (Pers.) Rostr.	*Ust. tritici* (Pers.) Jens.	Internally seed-borne, (floral infection.)
	Flag smut . .	*Urocystis tritici* Koern.	No change	Externally seed-borne.
	Karnal bunt . .	*Neovossia indica* (Mitra) Mundkur.	*Til. indica* Mitra . .	Air-borne, floral infection.
	Rough-spored bunt.	*Tilletia caries* (DC.) Tul.	*Til. Tritici* (Bjerk.) Wint.	Externally seed-borne.
	Smooth-spored bunt.	*Tilletia foetida* (Wallr.) Liro.	*Til. foetans* (*B. & C.*) Trel. or *Til. levis* Kuehn.	Externally seed-borne.
Rice (*Oryza sativa*)	Leaf smut . .	*Entyloma oryzae* Syd.	No change	Not known.

	Bunt	*Neovossia horrida* (Tak.) Padw. and Azmt.	*Til. horrida* Tak.	Probably air-borne (floral infection.)
Jowar (*Sorghum vulgare*).	Grain or covered smut ..	*Sphacelotheca sorghi* (Link) Clint.	No change	Externally seed-borne.
	Loose smut ..	*Sphacelotheca cruenta* (Kuehn) Potter.	No change ..	Externally seed-borne.
	Long smut ..	*Tolyposporium ehrenbergii* (Kuehn) Pat.	*Toly. filiferum* Busse.	Probably air-borne (floral infection.)
	Head smut ..	*Sphacelotheca reiliana* (Kuehn) Clint.	*Sorosporium reilianum.*	Probably soil-borne.
Oats (*Avena spp.*)	Loose smut ..	*Ustilago avenae* (Pers.) Rostr.	*Ust. avenae* (Pers.) Jens.	Externally seed-borne.
	Covered smut ..	*Ustilago Kolleri* Wille	*Ust. levis* (Keller. and Sw.) Magn.	Externally seed-borne.
Barley (*Hordeum* spp.)	Loose smut ..	*Ustilago nuda* (Jens.) Rostr.	*Ust. nuda* (Jens.) Kell. and Sw.	Internally seed-borne (floral infection.)
	Covered smut ..	*Ustilago hordei* (Pers.) Lagerh.	*Ust. hordei* (Pers.) Kell. and Sw.	Externally seed-borne.

Crop	Common name of smut	Scientific name according		Mode of transmission
		International Rules of Botanical Nomenclature	Previous practice	
Maize (*Zea mays*)	Smut	*Ustilago mays-zeae* (DC.) Corda.	*Ust. zeae* (Beckm.) Unger.	Air-borne.
	Head smut . .	*Sphacelotheca reiliana* (Kuehn) Clint.	*Sorosporium reilianum* (Kuehn) McAlp.	Not known.
Bajra *Pennisetum typhoides.*)	Smut	*Tolyposporium penicillariae* Bref.	No change	Air-borne.
	African smut . .	*Tolyposporium senegalense* Speg.	No change	Unknown.
	Bunt	*Tilletia ajrekari* Mundkur.	No change	Probably air-borne
Kagni or rale (*Setaria italica.*)	Grain smut . .	*Ustilago crameri* Koern.	No change	Externally seed-borne.
Chena (*Panicum miliaceum.*)	Head smut . .	*Sphacelotheca destruens* (Schc.) Stev. and John.	*Ustilago panicimiliacei* (Pers.) Wint.	Externally seed-borne.

Sawan (*Echinochloa frumentacea.*)	Rough-spored grain smut.	*Ustilago panicifrumentacei* Bref.	No change	Not known.
	Smooth-spored grain smut.	*Ustilago paradoxa.* Syd. and Butler.	No change	Externally seed-borne.
	Inflorescence smut	*Ustilago crus-galli* Tracy and Earle.	..	Not known.
Ragi or Nachni (*Eleusine coracana.*)	Grain smut.	*Melanopsichium eleusinis* (Kulk.) Mundkur and Thirumalachar.	*Ust. eleusinis* Kulkarni.	Probably air-borne.
Kodra (*Paspalum scrobiculatum*)	Head smut	*Sorosporium paspali* McAlp.	No change	Externally seed-borne.
Kasi (*Coix lacrymajobi*)	Grain smut	*Ustilago coicis* Bref.	No change	Not known.
	Smooth spored grain smut.	*Ustilago lachrymajobi* Mundkur.	..	Not known.
Sugarcane (*Saccharum* spp.)	Stem smut	*Ustilago scitaminea* Syd.	*Ust. sacchari* Rabenh.	Through infected setts and nodes
	Covered smut	*Sphacelotheca sacchari* (Rabenh.) Cif.	*Ust. sacchari* Rabenh.	Not known.
Mustard (*Brassica* spp.)	Root-gall smut	*Urocystis brassicae* Mundkur.	*Uro. coralloides* Rostr.	Soil-borne.

SUMMARY

A list giving the common names of smuts, their scientific names in conformity with the International Rules of Botanical Nomenclature and names according to previous practice, together with the probable modes of their transmission, has been compiled so as to standardize the names.

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CHAPTER VIII

THE MORE COMMON DISEASES OF THE POTATO, TOMATO, CHILLIES, BRINJAL AND TOBACCO IN NORTHERN INDIA

The Common Diseases of the Potato in Northern India

The potato is one of the more important food crops of India and is grown in every district. It is subject to many diseases but there are a few that are more serious than others. In the areas where the temperature goes below 70°F. during the growing season, late blight is serious. It is not of importance on the plains except during very favourable seasons. Early blight is common on the plains and sometimes causes serious losses by defoliating the vines before the tubers are mature. The virus diseases are also serious. There are not many of the viruses found in India but they cause serious losses. Root rot diseases are among the most serious and universally distributed. Among the fungi and bacteria that attack the roots and stems under ground are *Rhizoctonia, Macrophomina, Fusarium, Verticillium, Bacterium Solanacearum, Bacterium carotovorus, Sclerotium* and others. This is an imposing list and it is no more imposing than the damage done by the various ones, either singly or combined into groups as the case may be:

Phytophthora Late Blight of Potato

Hosts. It has been observed on all of the old cultivated varieties of *Solanum tuberosum* as well as varieties of tomato, *Lycopersicon esculentum* (96).

Geographical distribution. It is world-wide at present, although it is evident from recent findings that it was first in the New World (104). At present the fungus is reported from every section where potatoes are grown.

Appearance on the host plant. The first signs of the disease appear as small brown patches on the leaves. If the conditions are ideal for the spread of the fungus it rapidly spreads from leaf to leaf and over the same leaf. The whole surface may be involved. The rate of advance of the disease is controlled by the environmental conditions. When the days are clear and dry the spread of the disease is slow, but when the days are humid and the temperature is optimum the spread is rapid, from two to four days being all that is necessary to kill the entire leaf.

Infected leaves rapidly turn black and the tissues give off a smell of rotted vegetable matter. As the disease advances the whole plant may become blackened and rotted. Under favourable weather conditions a crop may be wiped out in a few days or weeks. Most of the decay which is noted is due to secondary fungi and bacteria which attack the weakened tissues.

If the blighted leaves are examined they will be found to have a delicate whitish growth over the under surface and, upon examination with a lens, it will be seen to consist of many branched conidiophores. These emerge in clusters from the stomata and, in the case of the potato, are found mostly on the under surface of the leaf. This whitish growth is rarely seen, or, if present, is very scanty if the weather is dry, but when the days are damp and cloudy the growth may be so rank that the surface of the leaf has a faint cottony appearance. On the tips of the conidiophores are borne somewhat lemon-shaped conidia which are readily scattered by wind, rain, insects, birds, tools and animals.

The fungus also attacks the tubers. In some cases the attack seems to be confined to the tubers. Butler (93) refers to the disease in Australia as often being found only on the underground parts, rarely showing on the upper portions. The effect on the tubers is to cause a dry rot which may not soften the tissues but causes rusty brown markings just below the surface of the skin. If, however, the conditions of the soil are such as to permit wet rot, it may be that the tubers decay with a wet rotting with considerable leaking of water. The affected portions may remain nearly normal but usually they are somewhat sunken and slightly discoloured over the affected area. When the seed piece is diseased the fungus may not appear above the ground but remain confined to the tubers, passing from the diseased seed piece to the tubers by way of the stolons. It often happens that diseased tubers will rot in storage and the losses at that time may be much greater than in the field.

The organism. *Phytophthora infestans* (Mont.) de Bary. *Botrytis infestans*, Montague.

Life cycle. The fungus probably over-winters as oospores in the soil or in the refuse from the previous year's crop. So far there is no very authentic evidence that the fungus can live saprophytically as mycelium. Oospores in diseased plant parts and tubers and the planting of diseased tubers are the two most probable sources of infection, although the part played by the oospores has not been definitely proved.

Most of the evidence points to the hibernation of the mycelium in the potato tubers and as the tuber is planted the fungus spreads from the seed piece to the stems and leaves on the one hand and to the tubers and stolons on the other. Once the fungus has gained the stems and leaves it soon produces the characteristic symptoms and the conidiophores and conidia rapidly develop. The conidiophores are slender branched structures which arise directly from the internal mycelium and emerge through the stomata. The conidiophore is usually about 10 microns in diameter and divided into from two to four branches of varying length. The conidia are borne on the tips of the conidiophore branches and as they mature the branch continues to elongate but the spore is pushed to one side and soon falls off.

The conidia are multinucleate, ovoid to lemon-shaped and measure from 22—32 by 16—24 microns. They are sometmies called sporangia as they contain from 7 to 30 nuclei which become zoospores and, as the sporangium (conidium) matures and ruptures, emerge as typical zoospores with two flagella. The zoospores swim rapidly about and, depending upon the temperature, in the course of time settle down, lose their flagella and become inclosed in a wall resembling that of the sporangium from which they came. They germinate by means of a germ tube and, if in the environment of a susceptible host penetrate into the tissues and the invasion begins.

During the season the spread of the disease is by means of the conidia which are spread about by wind, rain, insects, birds, animals and the tools and person of the cultivator. The temperature is a very important factor in the spread of the disease. Melhus (479) found that the optimum temperature for germination lies between 10 and 14 degrees C. It has been determined that at 24 degrees C. there is more direct germination by means of a germ tube, whereas at the lower temperatures there is more indirect germination by means of zoospores. Crosier stated the most rapid infection by zoospores was at 12-13 degrees C. If the temperature rises above 75 degrees F. for a short time the disease is checked and in those regions where the temperature is likely to rise above that point there is little damage from the disease. For that reason the disease is of little importance on the Gangetic plains but is found most destructive in the hill countries of northern India and Assam.

Control. Sen (679) reports control of the disease in Shillong by the use of Bordeaux mixture. The early varieties were most affected. Ramsey states that in the field the use of copper dusts has been found effective and that a 1 : 300 formaldehyde dip has been useful in preventing decay.

In recent years there has been an interest in the use of resistant varieties. In Europe and the United States there have been some very promising hybrids and selections found. Muller (506) tested some 115 varieties of potatoes and made crosses between those of South America and those of Europe. After testing some 15,000 plants over a period of three years he found that six varieties were virtually immune. When he studied the inheritance it was found to be due to four pairs of characters. Solaman (720) also reported that there are four pairs of factors governing inheritance of resistance to late blight. He found that resistance appeared to be about 1 : 300 among the plants he worked with.

Recently there has been work in the direction of selfing, or crossing and then back-crossing to increase resistance. Stevenson (750) reports the securing of resistance among strains in the United States by crossing two susceptible varieties. He also selfed a susceptible variety. From the cross of the two susceptible varieties a hybrid was secured that was only slightly injured by the late blight fungus. Stevenson (750) reports a variety called "Sibago" which was the result of a cross between two susceptible varieties that is moderately resistant to late blight and is superior in quality. It is also resistant to mild mosaic. From the results to date it is quite evident that the best road to control of such fungi as late blight is by way of plant breeding.

Reddick (648) reported that at Cornell University, U. S. A. a cross has been made between a South American selection and a commercial form which gives great promise. The South American variety was worthless commercially but highly resistant to late blight.

The cross appears to be resistant and carry the factors for tuber excellence as well. The most promising has been named the Ashworth, which is the name of the farmer in New York State who supplied the South American variety for crossing. Other promising varieties bear similar names. At present tests are being made to determine whether the new selections will maintain their resistance in other sections of the United States and of the world.

The Early Blight of Potatoes

Host plants. Potatoes, tomatoes, eggplant and related wild hosts.

Geographic distribution. World-wide.

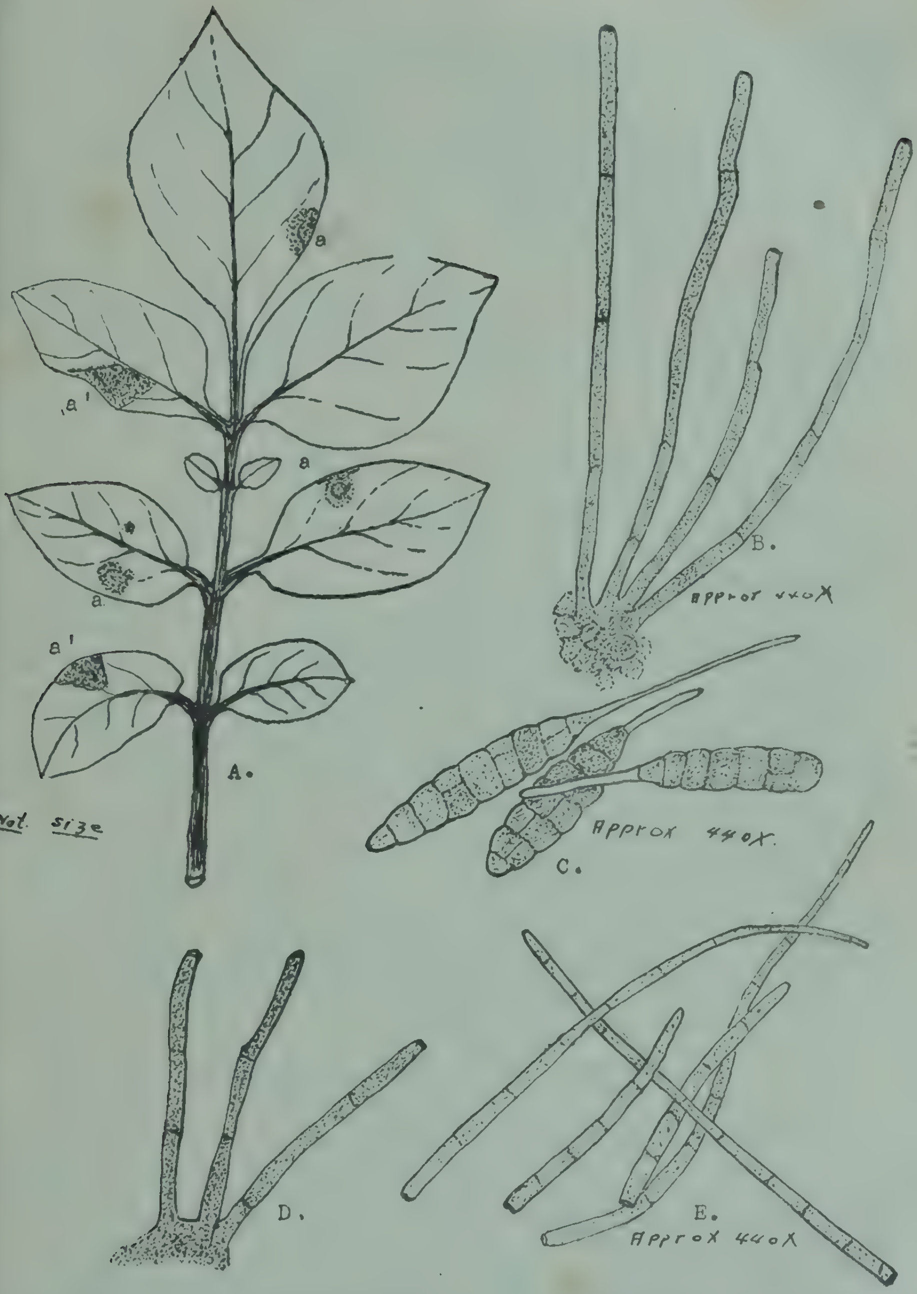
Appearance on the host plant. On the leaves the spots have been likened to the eye of the frog and called "frog eye" from the concentric rings of dark and light coloured areas. The fungus is confined largely to the leaves and it is there that most of the damage is done. Butler (93) stated that a tuber rot resulted from infection on those tubers which grew near the surface and were exposed.

Where the disease is severe, the entire top may be covered with the spots and it may result in entire fields being a mass of brown dead tops. One field on the Institute farm at Allahabad was so severely attacked in 1938 that scarcely any green could be seen.

The organism. Alternaria solani (E. & M.) Jones and Grant.

Because it does not produce its spores in chains, it has been argued that the fungus should be called *Macrosporium* rather than *Alternaria.* But common usage has established the name *Alternaria* and it will probably continue by that name.

The conidiophores are ascending from a light brown, septate mycelium which is intercellular. On the dead areas under humid conditions conidiophores



Early blight, *Alternaria solani,* and *Cercospora* leaf spot, caused by *Cercospora concors,* on potato leaf. Note the ring-like arrangement of the clusters of conidiophores in the case of early blight and the absence of any rings in the *Cercospora* leaf spots.

A. a. Early blight. b. *Cercospora* infections.
B. Conidiophores of *Alternatia solani.*
C. Conidia of *A. solani.*
D. Conidiophores of *Cercospora concors.*
E. Conidia of *C. concors.*

arise in large numbers. They measure from 50 to 90 by 8 to 9 microns and bear a single conidium at the tip. The conidia are club-shaped with from 5 to 10 cross septa with few, if any, longitudinal septa. It is held that this is another reason for doubting the fungus being an *Alternaria*. The conidia measure from 120 to 296 by 12 to 20 microns. Up to the present time no perfect stage has ever been found. The fungus is rather unstable and saltations have been reported by several workers.

The fungus has not been said to cause much storage tuber rot of potatoes on the plains of India because of the heat which kills the spores. One wonders at this because it is evident that the spores do live over on the old refuse or on alternate hosts.

Control. Destroy all old debris so that the pathogen will have no place to live over winter on. Rotation will also help. Spraying with Bordeaux 4-4-50 for the early applications and 6-6-50 for the late applications. Where this is done increases in yield of tubers and cash returns may exceed 50 per cent.

Narsimhan (539) found that 1 ℔. of calcium arsenate in 50 gallons water with lime casinate as a spreader is effective against *Alternaria Solani*. See Heald (275) Butler (93) and Owens (563) for more complete discussions of the disease.

Cercospora Leaf spot of Potatoes

Hosts. Potato

Geographic distribution. North America, Europe, Asia. In India it has been recorded in Bengal, Pusa and Poona.

Appearance on the host plant. The spots are rounded but rather indefinite in general shape. They are somewhat like those of early blight but lack the distinct concentric rings of conidiophores which mark the spots of early blight. See diagram. They are pale grayish above but light below.

The organism. *Cercospora concors* (Casp.) Sacc.

Conidiophores arise from the stomata either singly or in groups. They are brownish, erect, septate and measure 40—80 microns. The conidia are hyaline, produced apically, are quite variable in form, slightly curved, 1-6 septate and measure from 15—90×4—6 microns.

Control. The disease is not usually serious and the same control measures used against early blight will also control the leaf spot.

Virus Diseases of Potatoes

Although the potato has been one of the main crops of India for many years, having been introduced into the country over 100 years ago, little was written about the virus diseases before 1930. It is probable that the virus diseases were not present before that time, or if present, were not recognized. It was not until about 1920 that the true nature of the virus diseases on plants began to be suspected. Pushkarnath (620) states that the most important virus diseases of potato are leaf roll and mosaic in the hills and streak on up-to-date in the northern hills. These are being studied at this time in the Indian Agricultural Research Institute and the other stations over the northern part of India. They will be described somewhat in detail.

Leaf Roll

This virus disease is referred to by Smith (717) as Solanum Virus 11. It was called the leaf-rolling virus by Schultz and Folsom in 1923. But it is probable that the virus may cause different characters under different circumstances and thus make it very difficult to make positive identification.

Hosts. Potato and probably various others of the Solanaceae, including the tomato and pepper.

Geographic distribution. At least in India, United States and Europe.

Appearance on the host plant. A diffuse mottling of the leflets which, if taken alone, cannot be distinguished from that of rugose mosaic. The leaves generally show an upward rolling that is the distinguishing characteristic. The leaves are soft and droopy and resemble those of plants affected with *Rhizoctonia.* Different varieties differ in the distinctness of the varieties.

The organism. Leaf-rolling Mosaic Virus (Solanum Virus 11).

Little is known about the properties of the virus.

Control. Control of leaf roll is confined to roguing and securing seed from an area that is disease free.

Mosaic disease

This disease is also known as *Potato Virus* Y and will be discussed under that name.

Hosts. Potatoes, tobacco, Jimson weed, as well as others.

Geographical distribution. Similar to the *Leaf roll.*

Appearance on the host plant. The symptoms are varied but in a large number of varieties of potatoes there is a streak and necrosis which under some circumstances may appear like an attack by late blight. Vasudeva and Lal (880) give the chief varieties in India that are subject to infection as Gala, Majestic, President, Windsor Castle and Talisman. Other varieties were affected to a lesser extent. On the President variety the symptoms begin after some three weeks with a blotchy mottle spreading from the veins and affecting the top most leaves. Later a necrosis appears on the under side along the veins. Later the necrotic areas will appear on the upper surface and in time will pass down the petiole and reach the main stem. The leaves become necrotic, wither and droop so that they appear to hang by a very small part of the tissue. As new leaves come out they may not become necrotic but are mottled and crinkled. These characters are not constant and any one who expects to become efficient in identifying virus diseases cannot do it reading a book such as this or any other. It requires careful study in the field with regular observations.

The organism. Potato Virus Y Smith. (*Solanum Virus* 2 **Orton.**).

Control. Pal (592) in 1943, gave out data that indicated that the virus disease can be controlled by observing rather simple methods. The first step is to secure as nearly disease-free seed as possible. The second is roguing. In one experiment reported he gave the following data;

(a) Seed from rogued fields.
(b) Tubers from fields where no roguing was done.
(c) Tubers from known diseased fields.

Time of planting

	40 days after planting	70 days after planting	100 days after planting
(a)	5.2%	7.5%	9.4%
(b)	9.8%	16.8%	19.8%
(c)	11.1%	24.9%	28.2%

He also reported that the date of planting has a definite effect and offers the following data as evidence;

Date of Planting

	Sep. 15	Oct. 1	Oct. 15	Nov. 1
per cent of disease	31.3	35.6	4.9	7.3

From the above it appears that it should not be impossible to have reasonably virus free potatoes.

Streak.

This virus disease is known as *Solanum Virus* 4 Murphy. It appears most closely associated with Up-to-date potato variety and is sometimes called the *Up-to-date Streak.*

Hosts. It has been found widely distributed among the varieties of potatoes and also on such other plants as jimson weed and closely related *Solanaceae.*

Geographic distribution. North America, England, Europe and India at least.

Appearance on the host plant. For some time *Solanum Virus* 4 was found so closely associated with *Solanum Virus* 1 that they could not be readily separated. Later by securing a variety of potato resistant to *Solanum Virus* 1., it was possible to secure *Solanum Virus* 4 alone.

The disease gets its name from the pattern of the chlorophyll mottling. The mottling being in streaks. But other symptoms are, necrotic spots on the topmost leaves and, in the case of susceptible varieties, the killing of the terminal buds. The President variety, previously mentioned, is one of the susceptible varieties.

The organism. Streak. Up-to-Date Streak Murphy. *Potato Virus B* Bowden.

Dilution of 1 : 1000, aging for weeks or heating to 70°C. for 10 mintes will destroy its virulence.

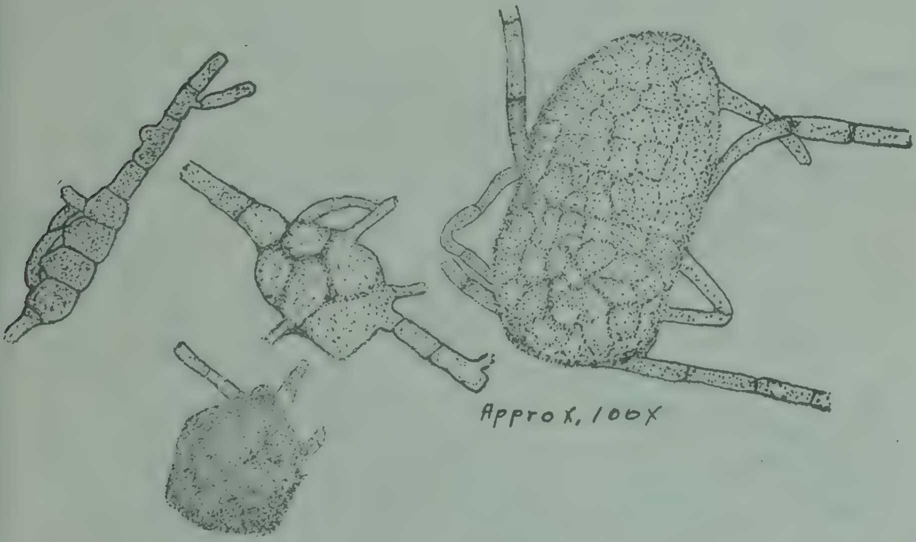
Control. The same measures as suggested for Mosaic are also recommended for control of Streak. Pal (592) suggests that a seed certification service be set up to help the growers in securing disease-free seed.

The Rhizactonia Disease

Host plants. The fungus is usually thought of as causing a disease of the potato but it has a wide range of host plants. Ajrekar (10) found some 21 different species of plants being attacked by the fungus in the neighbourhood of some infected mustard plants. It is especially bad on crucifers and potatoes and at times causes serious losses to cereals by producing a foot rot.

Geographic distribution. World-wide.

Appearance on the host plant. On potatoes it is commonly referred to as "the dirt that won't wash off". This refers to the black bodies that form on the outside of the potato tuber and become conspicuous when the dirt is washed off so that they show. On the young potato plants the fungus causes dark elongated areas on the stems. They may remain localized or girdle the stem, in which case the stem will wilt and die. The tubers may be rotted or show little external evidence of the fungus.



Rhizoctonia sclerotia from oat meal agar. Rhizoctonia species cause losses to the potato crop of 5 to 6 per cent yearly on the Agricultural Institute Farm. Treatment with Spergon has reduced, but not completely controlled, the fungus.

On crucifers it causes a disease often referred to as wire stem. The stems are shrunken and destroyed. In the case of cabbage, the fungus may progress up to the base of the head and destroy the vascular system and even the entire stem. Later the head itself may also be involved.

On wheat it causes a blight. The infected plants lose their green colour, wither and turn brown.

The organism. Rhizoctonia solani Kuhn. It is also known as *Hypochnis solani* Pril. & Del. The perfect stage is known as *Corticium vagum* B. & C. and is a *Basidiomycete.* Stevens (741) and Clements and Shear (128) place the genus *Corticium* under the *Telephoraceae* of the *Agaricales*, whereas Gaumann and Dodge (243) place it under the *Corticiaceae* of the *Polyporiales.*

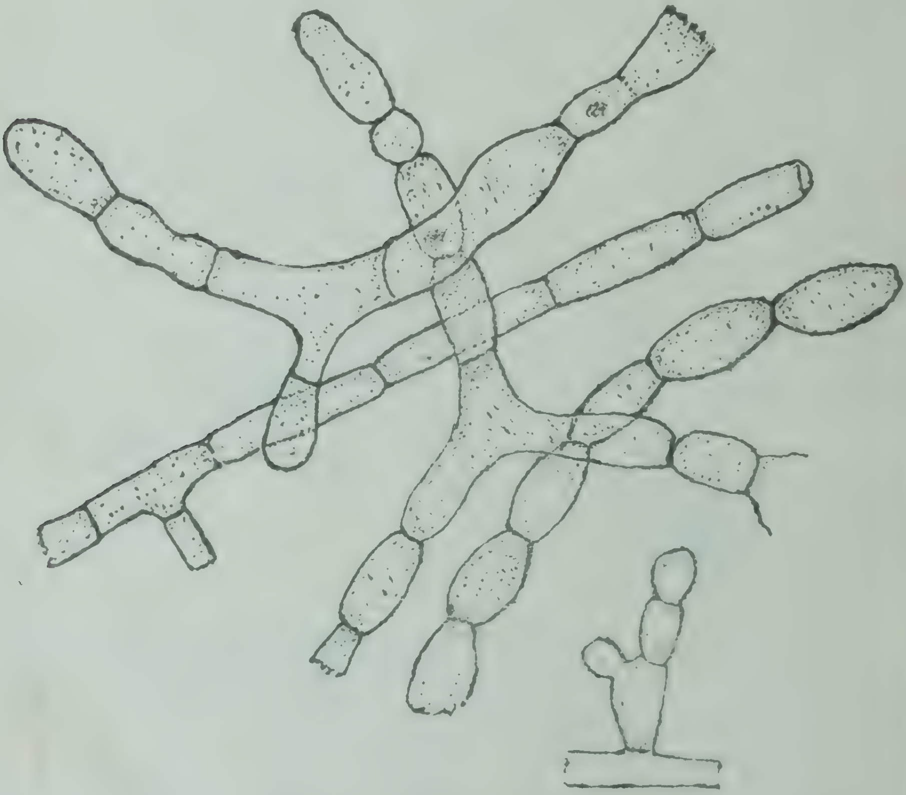


Diagram of mycelium of *Rhizoctonia solani* grown on oat meal agar.

The young vegetative hyphae are colourless, vacuolate, sparingly septate (100 to 200 microns) and show constrictions at the points of emergence of the branches. At the beginning of sclerotial formation the hyphae become more separate, the cells shorter and more barrel-shaped. This occurs when the vegetative growth ceases with the death and drying of the host tissues.

Sclerotial formation consists of repeated branching of hyphae to form short cells which adhere and finally form a mass of parenchymatous tissue. A cross section of a sclerotium will show the central cells large and thin-walled. The mature sclerotia measure 100 to 150 microns. They are produced most abundantly on the tubers and vary from one to many. Sometimes they may reach a diameter of an inch or more but usually are smaller than a grain of wheat. Most of them are on the surface of the skin but they have been reported within the tissues.

When the conditions are optimum the hyphae send up short branches which function as basidia and bear, typically, 4 basidiospores. For this reason it was placed in the genus *Hypochnis* by Prillieux and Delacroix. In America it is generally referred to the genus *Corticium*. The basidial stage is common under certain conditions and no doubt it is responsible for some of the widespread distribution.

Butler (93) describes *Rhizoctonia solani* on cowpeas and potatoes in India. He states that a second similar form was found which was slightly larger and which he considered to be *Hypochnis solani* Pril. & Del.

Shaw and Ajrekar (585) found a *Rhizoctonia* on potatoes which they considered to be *R. destruens* Tass. They also found the same one on *Piper betle* in Bengal and considered it the same one as found on *Delphinium*. They also found the fungus on several other plants including *Medicago sativa, Arachis hypogaea* and *Oryza sativa.* Other species on gram was identified *R. napi* West.

Mundkur (614) found a species of *Rhizoctonia* on sweet potatoes in a roadside market in the Bombay Presidency. He identified the fungus as *Rhizoctonia solani* Kuhn (*Corticium Vagum* B. & C.)

Control. Absolute control is costly and difficult. But there are several measures which will reduce the disease both in the field and among market produce.

Seed selection. In fungus free soil this is essential and in diseased soil it lessens the amount of infection.

Seed disinfection. Liquid dips, such as the cold and hot mercuric chloride and the hot formaldehyde are effective. Organic mercury dusts are also recommended.

Rotation and cultural practices. The fungus does best in low temperature and if planting occurs during the cooler portion of the season there is likelihood of more infection. In the United States they have found that banking the soil up against the stem will increase the diseases because the sclerotia are placed in contact with the stems.

Macrophomina phaseoli or Charcoal Rot on Potatoes

Hosts. Practically all of the cultivated crops.

Geographic distribution. World-wide.

Appearance on the host plant. The tops may not show the early symptoms of the disease. Later they turn yellow, wilt and die. The attack on the tubers usually comes at the beginning of hot weather and when the plant is maturing. Pathogen appears to enter by way of the seed piece and then go to the roots. Infections of the tuber is by way of the stolon and the eyes. The infection is probably not very deep and, if other organisms do not enter in, the tuber will probably dry down and rotting stop. This is not often the case, however, as other organisms usually enter in and soft rots follow.

The symptoms on the tuber are some what darkened areas that appear softer than the rest of the tuber and lightly water soaked. See photograph. This area is not usually very deep.

The organism is usually associated with such fungi as *Bacterium carotovora, Sclerotium rolfsii* and *Fusarium* species.

Storage rotting is largely due to field infections and the leakage in the pits or bins is due to the secondary organisms. *Rhizoctonia solani, Phytophthora infestans* and *Bacterium solanacearum* are also associated with the root and tuber rots.

The organism. Macrophomina phaseoli (Maubl.) Ashby *Sclerotium bataticola* Taub.

On potatoes it is the sclerotium stage that is most commonly found. It is recognized by the type of infection (See photograph) and by the sclerotia (See diagrams). . These will be small, however, and may not always be present especially on the newly infected tubers. Isolation is easy and is the surest way of identification.

The organism is potentially a high temperature organism. Rotting is accelerated in high temperatures. For that reason it is essential that the temperature of the storage pits be kept as low as possible.

Control. Rotation is of little value. Early harvesting before the weather becomes too hot and before the tubers are completely mature will reduce the amount of infection harvested. Use well drained land. Sort out the diseased tubers immediately at harvest time and remove them. Dastur (155) recorded *Sclerotium bataticola* and *Fusarium* spp. as serious on stored potatoes. He states that the best method of keeping them is in pits 24 to 30 inches deep, lined with dry leaves and ventilated with hollow bamboo stems. These should be surrounded by a trench some 6 inches deep and 4 inches wide.

Sclerotium rolfsii on Potatoes

Hosts. A wide range of host plants. Butler and Bisby (96) list some twelve including a number of legumes common on the farms.

Geographic distribution. World-wide. Butler and Bisby (96) record it from all parts of India.

Appearance on the host plants. Yellowing wilting of the foliage is a common symptom of the fungus attack. But as this is also common as a symptom of several other fungi as well, examination of the root system is necessary before positive identification can be made. On the roots and tubers of infected plants the small mustard seed like sclerotia make identification easy. Without the sclerotia the fungus may be confused with species of *Fusarium* which cause dry rot of the tubers. The mycelium of S. *rolfsii* is less abundant than that of the *Fusarium* species.

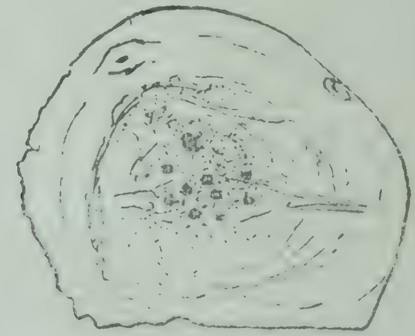


Diagram illustrating a partly mummified potato showing the sclerotia of *Sclerotium rolfsii.* At Allahabad this fungus causes slight damage in the field and in storage.

In India the fungus may cause damping off of small seedlings. It was isolated from cabbage seedlings on the Agricultural Institute farm which were damping off in the seed bed. Ramakrishnan (636) successfully inoculated potato seedlings with a strain of S. *rolfsii* isolated from Zinnia. Bertus (57) stated that S. *rolfsii* could cause damping off of the potato seedlings. Pearl

(602) stated that the fungus caused a serious root and tuber rot in the C. P.

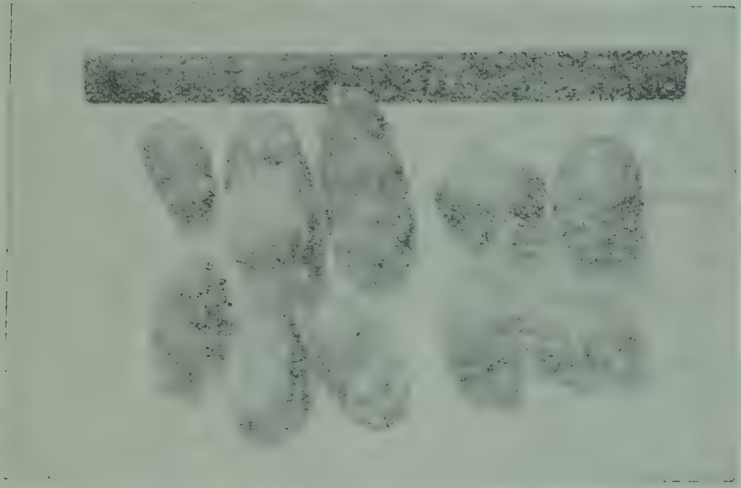


Photo of potato tubers infected with *Sclerotium rolfsii* and normal healthy ones. Diseased tubers on the left. Healthy tubers on the right. (Photo by Dr. W. N. Rice).

The organism. *Sclerotium rolfsii* Sacc. Perfect stage *Corticium rolfsii* Curzi.

The sclerotia are small, brown mustard-seed-like bodies borne on the whitish, aggressive mycelium. It grows both saprophytically and parasitically.

Control. The fungus grows more vigorously in damp soil than in dry. Then drainage is one step to follow in control. Seed disinfection also a method. Uppal (849) stated that Kerol used at 1 : 1200 as a spray at 30-day intervals controlled without plant injury. Rotation may be followed. Last year a small percentage of infection was observed at Allahabad.

Verticillium Wilt of Potato

Hosts. Wide range of hosts.

Geographic distribution. World-wide.

Appearance on the host plant. Although it is referred to as wilting, actually the wilting is not as common as other symptoms, such as a yellowing of the leaves, a browning and discolouration followed by death of the plant.

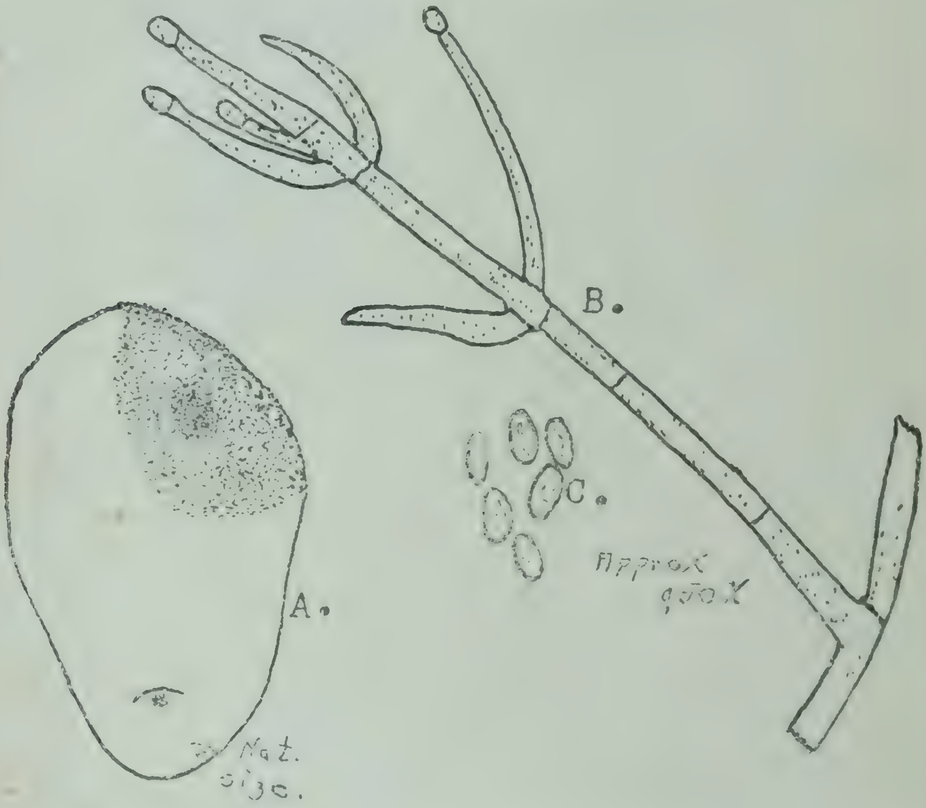


Diagram illustrating potato tuber infection with *Verticillium*.
- A. Tuber showing decayed area.
- B. Conidiophore.
- C. Conidia.

The organism. The fungus possesses a creeping hyphae that resembles that of *Fusarium* somewhat in microscopic appearance. The conidiophores are erect, branched (see drawing), septate and hyaline. The branching is more or less dichotomous, or even trichotomous. Conidia single, round, elliptical, ovate, hyaline or slightly coloured.

Control. Sanitation. Remove and burn the old dead trash from the previous crop. Check the host range and do not rotate them. Cereals are not susceptible so that they may be included in a rotation.

Bacterial Wilt or Ring Disease of Potato

Host plants. This disease has one of the widest ranges of any bacterial disease found in India. It has been reported on at least 117 species (207) scattered through 73 genera and 21 families. The range extends from the *Commelinaceae* to the *Compositae,* being most important on the *Solanaceae.* Butler (93) states that it was first reported in detail in 1909 in Mysore but that it was known much earlier than that. It had been of importance in Bombay for some years after 1891. In Bengal it has been the cause of a serious tobacco wilt.

Geographic distribution. It is practically worldwide in its distribution. It has been reported in the tobacco and potato-growing sections of the United States, in a large number of the South American countries, most of the European countries. many of the islands, such as the Dutch East Indies, New Zealand, Australia, the Philippines, Japan, in China, India and so on.

Appearance on the host plant. The disease is readily identified by the rapid wilting of the plant with the flaccid appearance of the leaves. It usually affects the lower leaves first but soon spreads to others and then the whole plant. The name "ring disease" comes from the appearance of the tubers which show a black ring just beneath the corky layer. This is due to the invasion of the vascular tissue by the bacteria.

Other names given to the disease are "Granville wilt", "Slime disease", "brown rot of *Solanaceae*", "ring disease of bangdi" and "bangle disease", as well as a number of other terms which are more or less descriptive.

From the vascular ring the bacteria invade the rest of the tissue and the whole potato becomes a soft rotten mass. Infection takes place by way of the stolon. In the case of tobacco and potato leaves, the veins may be brown. In the case of stems of tobacco and the tomato the pith may be destroyed and extensive cavities be formed.

Organism. The term *Bacterium solanacearum* (E. F. Smith). E. F. Smith, appears to be the correct one at this time. It was named *Phytomonas solanaceara* (E. F. Smith) Bergey et al., 1923, but as this name is not permitted, (208) the term *Bacterium* appears best. It was named *Bacillus solanacearum* E. F. Smith in 1895. Then in 1904 it was named *Bacillus nicotianae* by Uyeda. In 1906 it was named *Pseudomonas sesami* by Malkoff and in 1911 it was called *Bacillus musae* by Rorer. E. F. Smith renamed it *Pseudomonas solanacearum* in 1914, hence the name as it appears above.

It is motile by one polar flagellum measuring 0.5 × 1.5 microns in size. It does not produce capsules or spores. It is a Gram-negative, aerobic organism which reduces nitrates but produces no acid or gas. On agar, the colonies are small, irregular, roundish, smooth, wet, white or pale yellowish-brown. The optimum temperature is 35-37°C. The thermal death point is about 52°C.

Control. As the disease attacks the tubers of potatoes, it would follow that it might be planted with the tubers and that therefore care should be exercised to plant clean seed. Corrosive sublimate has been used for potatoes with success. For this purpose the usual formula is mercuric bichloride 4 ounces in 30 gallons of water. Young plants may be sprayed with Bordeaux and the disease held in check. One of the first things which the cultivator should do is to remove and destroy the leaves and stems of diseased plants. This should be followed by a rotation of crops which are affected by the disease. Even with these measures, however, at times the disease is not as easily controlled as might be expected. Park (596) reported that in tomatoes and eggplant the disease had become a limiting factor in sections of Ceylon as late as 1929.

Surayanarayana (768) states that if the cut potato seed is planted in the soil for a matter of 24 hours the infected pieces will begin to soften and show the rot whereas the normal healthy ones will begin to heal over the cuts. In this way the diseased may be detected and removed so that disease-free seed may be planted.

Fusarium Rot of Potatoes

Hosts. A wide range of host plants which includes the potato and a number of other plants of economic importance.

Geographic distribution. World-wide.

Appearance on the host plant. Among the first symptoms to be observed will be yellowing of the lower leaves and stems. The upper leaves may become yellow, wilt and finally die. The whole plant usually dies. There are a number of species of *Fusarium* which attack the potato and the symptoms of each are not all the same. *Fusarium oxysporium* attacks the tubers and will cause a dry rot which is not evident in the tops. There are exceptions, however, as Sen (679) in 1930 reported a serious wilting caused by *F. Oxysporium* in the Surma Valley, Assam. Infections occur mostly on the tubers, roots and stems. Where roots and stems are attacked the vascular portions will be found discoloured and a microscopic examination will disclose the mycelium in the xylem vessels. These are often plugged and this causes the wilting.

Although the most common *Fusarium* tuber disease symptoms are dry rots, it may happen that two or more species may combine and the rapidity of the decay increases so that wet rotting results. In fact, under optimum conditions, all of the species of *Fusarium* will produce some wet rot. Wet rot has been observed when *F. oxysporium* and *F. trichothecioides* occur together.

The organism. The organisms found associated with the diseases of potatoes are as follows:

Fusarium oxysporium Schlecht

Conidia are from 0-5 septate (mostly 3-4) with chlamydospores present. The conidiophores are branched. The macroconidia are slightly curved and somewhat flat tipped. The microconidia are tapered toward the apex.

Other species

F. solani Wr.
F. trichothecioides Wr.
F. radicicola Wr.
F. udum (Berk.) Wr.
F. coeruleum (Lib.) Sacc.

These species have all been reported on potatoes in India. The species descriptions will be omitted except for the brief list given for *F. oxysporium* and the student is referred to such articles as Wollenweber and Reinking (938) Sherbakoff (690) Padwick (570, 572, 574, 576) and others. Species descriptions are so nearly alike that it is only by actual study in the field and laboratory that one is able to distinguish them apart. Species differ under different conditions and the student would need to grow them on standard media and under standard (common) conditions to be able to identify them. This is a task for the research worker not for the farmer. The farmer is interested in the control and it is not of importance to him which fungus is causing the trouble and from our present knowledge it does not appear that such a highly technical knowledge is necessary for control in the field.

Control. The control of Fusarium root rots has been one of the major problems in the production of potatoes for a long time. A wide range of the organism has made rotations difficult and often uneffective. Seed treatment has helped and in some cases it has appeared to have protected the crop from the attack of the fungi of the soil. Spergon treated potatoes on the Institute farm actually had more diseased tubers at time of harvest than untreated ones but the ratio of diseased to healthy was approximately directly correlated with the increased production. It being evident that within the limits of that experiment there was no protection to the new tubers as a result of the treatment of the seed potatoes. Yellow oxide of mercury was used as a dip (1 lb. to 15 gallons water) before cutting and protection was secured up to 97-99% (Amer. Potato Journal XIX, 2, 19-23, 1942).

Storage rots are also a serious matter and is responsible for much loss between digging and marketing of potatoes. Careful sorting of the tubers before storage will reduce the losses from rotting. But it is not always possible to detect the infected potatoes before storing them and this means regular careful inspection during the strong period. As the decaying tubers are observed they can be removed and, while this is added expense, it pays in the lower total rot.

Work is being done under the Indian Agricultural Research Institute which gives promise. In 1946(166) four selections of potatoes, cultures 296 and 394, Aya Papa and M. 09 were still free from infection from *F. solani* after a month of exposure to the fungus under optimum conditions. Other selections were only slightly infected.

Black Heart Disease of Potatoes

Hosts. Tubers of the potato and possibly other underground tubers and possibly roots.

Geographic distribution. Apparently wherever potatoes are grown.

Appearance on the host plant. Black heart is a breakdown of the central tissues caused by storage at high temperatures in low oxygen. Bennett and Bartholomew (53) in 1924 placed tubers in closed jars at temperatures of 20 to 30°C. and others at 38°C. in free air. They found injury to the tubers in the closed jars after all the free air had been consumed. Those in the free air at 38°C. did not develop black heart. Davis (174) in 1926 found that a temperature of 45°C. will induce the disease. He concluded the breakdown of tissues is an O_2—CO_2 relationship. When the relationship of CO_2—O_2 was over 50 : 4 in the intercellular spaces there was an increased permeability of the protoplasm and death of the cells. Thus black heart appears to be a case of high respiratory activity and a failure of gas exchange to keep pace with respiration. The critical temperature appeared to be at 38°C. beyond which normal water relationships cannot be maintained. This was also reported by Mann and Joshi to be the critical temperature.

In 1938 Singh and Mathur (696) found that potatoes in the middle dormancy period would develope black heart within 48 hours if exposed to a temperature of 40.3 C. They found small active growing tubers more resistant than mature ones. Small ones remained unaffected after 105 hours at 57.1°C. Karmakar and Joshi (J. C. A. R. Bull. 45, 1941) in 1941 found the black heart would develop at 30°—32°C. after five months storage. When these were placed at higher temperatures they immediately rotted.

The organism. So far no organism has been shown to be associated with the disorder. There was one report from France that an organism was considered to be a casual factor but this appears to have been a mistake

Control. This appears to be largely a question of the proper relationship between ventilation and temperature. Low temperature with free air seems to be the best protection against the disease. The present average storage facilities are hardly adequate for the prevention of black heart on the plains but the storage house of the future must provide the optimum storage conditions for the tubers.

Potato Scab

Hosts. In India it has been reported on potato tubers only. Lutman (407), in 1945 has reported it on a number of crop, including Jerusalem artichoke, beets, carrots parsnips and others. Hooker and Kent (291) in 1946 infected radish seedlings in the laboratory and reported that the fungus killed the seedlings in the field.

Geographic distribution. Nandi (536) reported scab as serious on potatoes in Assam and Mitra (497) reported it on storage potatoes.

Appearance on the host plant. Scab appears in a number of different forms. It may be only a superficial browning of the skin with little abrasions. It may be a surface scab with more or less concentric rings about the centre. Sometimes the scabs may be raised so that they stand above the surface of the skin. Sometimes the scabs are pitted and deep rough walled depressions in the tuber. Sometimes there are small pimples like pustules which form on the potato tuber. It often happens that other fungi may also play a part and thus complicate the picture. Other organisms which cause disease sometimes referred to as scab, are *Synchytrichum endobioticum* causing potato wart; *Spondylocladium atrovirens* causing silver scurf; *Oospora pustulans* causing skin spot and *Spongospora subterranea* which caused powdery scab. At this time only the last has been reported in India.

The organism. Actinomyces scabies (Thax.) Gussow.

The organism is found in the order *Actinomycetales* which is just below the *Eubacteriaces* or true bactreia. They are between the true bacteria and the mould like forms. The mycelium is scanty and very fine. It was first described as *Oospora scabies* and some of the species are still called *Oospora.* See Heald (275) for a complete description. Recently Lutman (407) has given a very complete report of the work done on the disease.

Lutman (407) stated that the fungus is not distributed by the tubers as was once thought to be the case but is distributed by the old plant material and humus. It would seem to be a good saprophyte as well as a parasite. He suggests the removal of all old debris. This must be done thoroughly for the organism was readily isolated from old potato vines and humus after seven years. Lutman used a differential medium composed of pectin, arabinose and organic salts at a pH of 5.6 The pH. rendered the pectin unavailable except for a few bacteria. By using this form of medium he was able to secure isolations from the soil which had been kept in bottles for seven years. Humus appeared somewhat better than soil. Humus contained 46 per cent moisture whereas soil contained only 23 per cent moisture. When compost, containing old potato vines and parts of roots, was included in the potato beds the tubers were scabbed. This would show that rotation is not successful unless for a very long time. He contends that many thousands of rupees have been spent on seed treatment that has not been effective.

At a pH of 5.2 or below, it does not grow. Taking advantage of this weakness sulphur has been used and at the rate of 300—500 lbs. an acre which creates enough acidity so that the organism can be held in check. This is especially interesting because the disease actually increases from a pH of 8.5 to about 5.7 and then rapidly decreases to the 5.2 point when it can no longer grow.

THE COMMON DISEASES OF THE TOMATO IN NORTHERN INDIA

The tomato is one of the most common garden crops to be found in India although it was not a native of this land when man began to cultivate the soil. It is a native of Central and South America where it grows wild and where it was found by the Spanish explorers when they visited the New World. Carried to Europe it was grown along the Mediterranean coast for a number of years before it was considered to be good to eat. Just when it came to India is not known exactly but probably it was carried here by the Portuguese as was the case with so many other of the crop plants we know so well.

There are a number of diseases on the tomato which are serious. Perhaps the most serious disease here in India is the 'mosaic or the disease due to the viruses. Early blight is also present on the tomato, although not in as severe a form as on potato. Late blight is sometimes found. (For that see under potato) *Fusarium* wilt is also serious in many cases. Fruit rot, soft rot of fruit, damping-off and leaf mould are also important. There are a number of fungi that are found on the tomato roots causing rotting. Among them are *Rhizoctonia solani, Macrophomina phaseoli, Sclerotium rolfsii, Fusarium and Pythium* species. Most of these have been discussed in relation to potato diseases and will not be repeated here. Root rot control is best brought about by destruction of the old debris, rotation and the use of organic manures before planting.

Virus Diseases of the Tomato

At the present time there is a very brief literature on the virus diseases of tomatoes in India. It would appear that few of the viruses known over the world have been recognized here, or, if so, they have not been reported. Leaf roll of potato has been transmitted to tomato by Venkata Rao. In 1933 Dastur recorded spotted wilt on tomatoes in the C. P. and in 1934, Desai (180) described a virus disease on tomatoes that appears to be very similar to the Bushy Stunt Disease of Smith (717). Symptoms of the most common virus on tomatoes in the Allahabad region are very similar to the bushy stunt disease. The description of the two virus diseases mentioned will be given below.

Spotted Wilt Virus (*Lycopersicum Virus* 3. Brittlebank).

Hosts. Smith (717) lists over 100 different species of plants which have been recorded as hosts for the virus. This includes 19 families of the *Dicotoledonae* and 2 families of the *Monocotyledonae.*

Geographic distribution. Since the first report of the virus was made in Australia in 1915 it has been reported from every section of the world. It is not surprising when one considers the host range and the geographic distribution of the hosts.

Appearance on the host plant. Smith (717) gives the symptoms as a slight intensification of the vein thickening of the younger leaves, a tendency to curl downward of the younger leaves and then a very characteristic bronzing of the lamina between the veins. There may be a distinct yellowing of the leaves in a mosaic like mottling. Leaf distortion and some stunting may occur. Fruit symptoms, if any, are likely to be a spotting of the skin with lighter red, yellow or even white areas. If extensive it may cover the fruit in such a way that the normal red of the fruit may appear as little islands in a lighter coloured area.

The organism. Spotted Wilt. Lycopersicum Virus. Brittlebank.

Control. Out-doors the problem of control of the virus diseases is a very difficult one. Insect vectors are difficult to control and in many cases we may not know all of the vectors. It is known that thrips are among the most common of the vectors of *Lycopersicum Virus* 3. In some cases dusting with insecticides may help but most of the recommendations are for planting the tomatoes away from any of the known host plants, like chillies, tobacco, potato, petunia, Zinnia, ground cherry, Antirrhinum, common beans or peas, Nasturtium, cauliflower, poppy or peonies. That is only a small portion of the list but it is enough to indicate that tomatoes would have to really be *alone* if they are to be away from the vicinity of any other host plants. But at least it is essential that as far as possible there are no hosts very close at hand, such as chillies, tobacco or potatoes. There are a number of weed hosts as well. Among the weed hosts common to the fields are wild lettuce, bind-weed, wild cape gooseberry, and the nightshade (*Solanum nigrum*). These should be kept out of the area as they may harbour the virus and the insect vectors may carry it from them to the tomatoes.

Tomato Bushy Stunt

Host plants. While it is confined mostly to the family *Solanaceae,* this virus appears to have a number of hosts outside of the potato family.

Geographic distribution. It has not been officially recorded in many countries but from the descriptions it appears that it may be found in a number of them. Characters seen on the tomatoes on the Institute Farm, Allahabad are so nearly identical with those given for the *Tomato Bushy Stunt* that it is believed at this time the virus is here. More observations are needed and inoculation work done, however, before a positive statement can be made.

Appearance on the host plant. There is a definite stunting of the plants; the leaves are small and distorted, the terminal growing point appears diseased or destroyed and a number of secondary branches develop. They are small and also bear small, more or less distorted leaves. Flower buds rarely open normally but usually abort. Fruits are rare and they are small and light coloured if any develop. Stem of the diseased plants are usually weak and fall over, the ends of the branches curving upward and in some cases they appear almost brush-like. Such plants usually die early but they may remain in the field after the main crop is harvested.

The organism. Bushy Stunt Disease or *Lycopersicum Virus* 4.

Control. Removal of the diseased plants as soon as they are observed and their destruction. They should not be left in the field to act as sources of infection for the other plants.

Other virus diseases are common on the tomato. It is a host for nearly every known virus disease that is on the potato or tobacco. Many of the symptoms are difficult to recognize because they are a mixture of several reactions at the same time and only the expert can identify them. But the one thing the grower can do is to look for the typical leaf mottling, curling, dwarfing or other signs and remove that plant from the field as soon as possible. In this way he can reduce the danger of spread among the healthy plants.

Fruit Rot of Tomatoes

Hosts. The organisms causing the fruit rot of tomatoes vary in number and virulence and it is fairly certain that the tomato is not the only host plant attacked nor is the fruit the only part attacked. Isolations from rotting fruit gathered on the Agricultural Institute farm, Allahabad, proved to be a species of *Rhizoctonia, Pythium, Fusarium* and a number of fungi that appeared to be only saprophytes.

Geographic distribution. These species of fungi are known to be widely scattered.

Appearance on the host plant. The first symptom of the fruit decay was a water soaking of the portion touching the ground or another fruit which was decaying. The fruit rapidly becomes soft and watery. When the soft rot organism, *Bacterium carotovorus,* is involved the fruit rapidly collapses. The rate of collapse in the case of *Pythium* and *Rhizoctonia* rotting is much slower although it is more more rapid in the case of *Pythium* than of *Rhizoctonia.* When *Pythium* was concerned there was a felt of mycelium developed around the stem within a matter of 36 to 48 hours after infection took place. See illustration below.

The organisms. Rhizoctonia solani Kuehn (*Corticium vagum* B. & C.) *Pythium species.* The one associated with the soft rot of fruit in 1946-47 appeared more like *P. debaryanum* Hesse. Characteristics of the

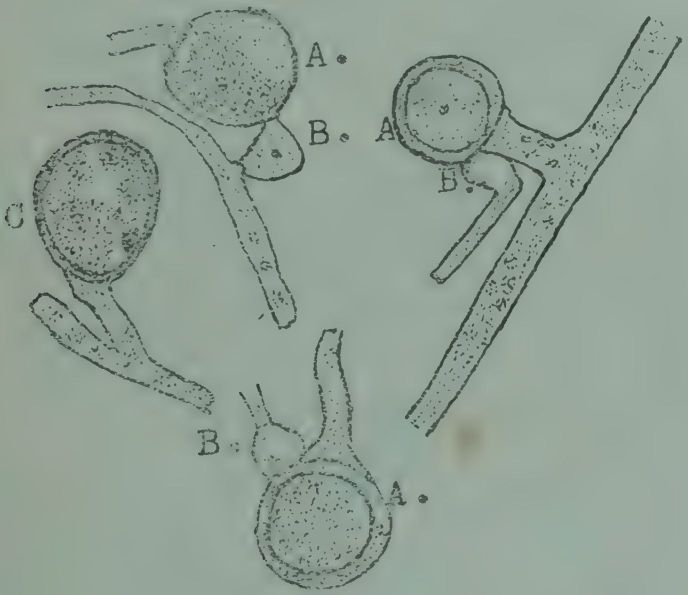


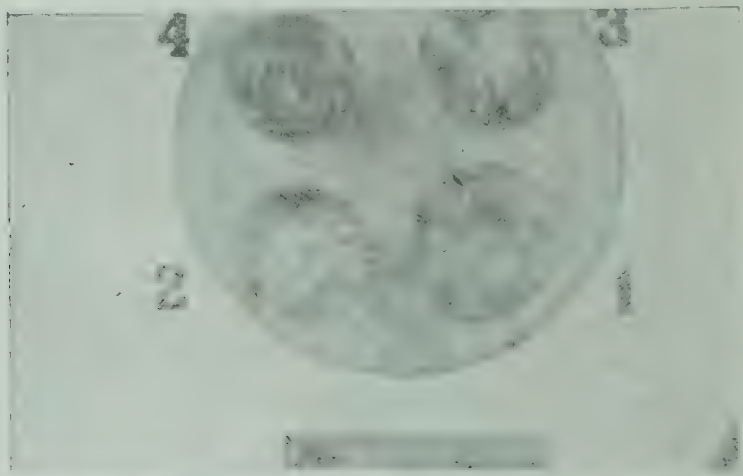
Diagram of a Pythium, isolated from rotting tomato fruit collected on the Institute Farm at Allahabad, which would pass through a 2 inch tomato fruit within 36 hours after inoculation.

A. Oogonia.
B. Antheridia.
C. Sporangium.

fungus, when grown on oat meal agar, are shown in the diagram.

Control. Observations and simple experiments soon demonstrated that the fungi were soil-borne and that if the fruits could be kept off the damp soil they would not rot. However, if a fruit of a cluster touched the soil and became infected the whole cluster would ultimately become decayed.

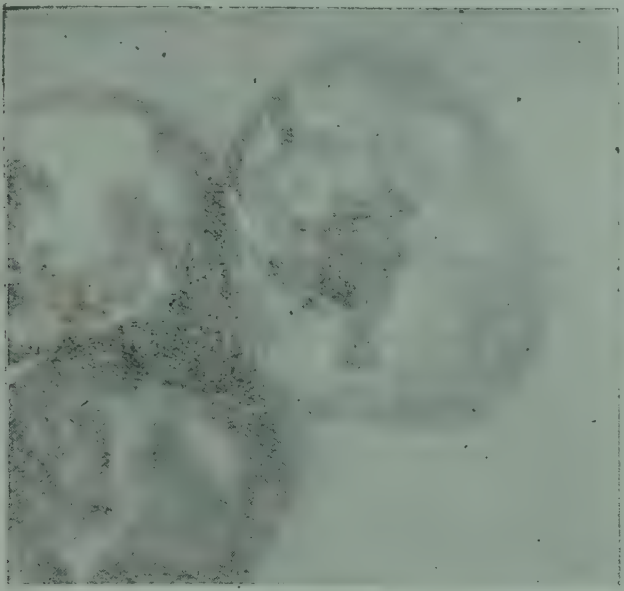
It was found that the *Pythium* was much more active as a fruit-rotting agent that the *Rhizoctonia.* When healthy green fruit was inoculated with a pure culture of the Pythium under laboratory conditions, it would pass through the fruit and form a felt of mycelium around the opposite end within 36 hours. *Rhizoctonia* required some four days to pass through a similar sized fruit inoculated in the same way with a pure culture of *Rhizoctonia.* When inoculations consisted of both fungi the *Pythium* would appear within



Photograph of tomato fruit illustrating the type of rot occurring when *Pythium* and *Rhizoctonia* are concerned.

1. Control.
2. Inoculated with *Pythium* alone.
3. Inoculated with *Pythium* and *Rhizoctonia.*
4. Inoculated with *Rhizoctonia* alone. (Photo by Dr. W. N. Rice). Taken 36 hours after inoculation.

some 36 hours but the *Rhizoctonia* was usually so slow that the whole fruit collapsed before it appeared.



Photograph illustrating the type of soft rot resulting from the tomato fruits touching the ground.

Control. Keeping the fruits from touching the soil appears to be the best means of control of the fruit rot. Controlling irrigation, where this is practised, may also help.

Early Blight on Tomato

The early blight of tomato is caused by the same fungus as causes the early blight of potato. Leaf characters are somewhat the same but on the tomato the fruit is also attacked with sometimes serious results.

Hosts. The same as for the potato organism.

Geographic distribution. World-wide.

Appearance on the host plant. It may be on the young seedlings as a stem canker or collar rot. It may appear on the young leaves and kill the lower leaves. On the older plants it does not do much damage except under very favourable conditions for the fungus. The first symptoms are small irregular brown spots that soon die in the centre. As they enlarge there may be concentric rings which vary in size, smaller ones will be a millimeter or so in diameter and the larger may be a centimeter across. These are often referred to as the frog-eye pattern which is quite similar to that of the early blight on potato. If there is high temperature and high humidity at the time of the attack the plants may be completely defoliated. At Allahabad in 1937, the fields were completely browned before harvest-time by the attack of the early blight fungus.

Stem lesions are small, dark, slightly sunken areas which may later enlarge to form more or less circular areas with light sunken centres. Stems so attacked are

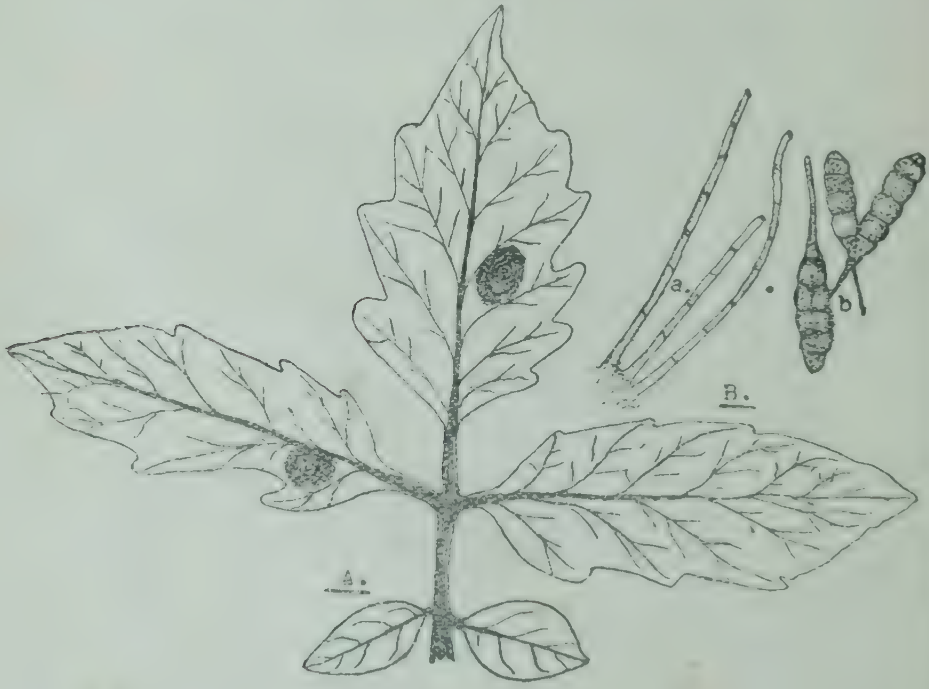


Diagram illustrating early blight on tomato leaves.
A. Leaf with infection areas.
B. a. Conidiophores of *Alternaria solani*. b. Conidia.

weak and may break at that point. They also act as points of infection if set out in the field. In the case of older plants the stem lesions are lighter in colour but with darker margins.

The organism. Alternaria solani (Ell. & Mart). Jones & Grout.

For the description see under Early Blight of Potato.

Control. The fungus is probably saprophytic to some extent and therefore much of the seedling infection may be from the soil. Crowding of the seedlings should be avoided. The seed should be treated with bichloride of mercury (1 : 800) or one of the mercury compounds as a protection from the fungus. In the seedling beds there should be good ventilation and heavy watering should be avoided at all times. If there is evidence of the fungus in the seed beds Bordeaux mixture may be used (2-2-50) but should not be used for at least a weak before transplanting as the plants are likely to wilt from the effects of the Bordeaux. After the plants are well established in the field they may be sprayed with 4-4-50 Bordeaux without damage.

Fields known to be infected should not be used and the seed beds should be changed frequently.

Fusarium Wilt of Tomatoes

Host plants. Tomatoes and closely related crops.

Geographic distribution. It is common where tomatoes are grown. It was reported to be serious in New South Wales in 1938, (Ag. Gaz. N. S. W. I. pp. 367-371, 1939).

Appearance on the host plant. The disease does not usually appear until the fruit begins to set. Wilting may be noticed at that time, although some plants may die before blossoming time. Here and there one may find plants that appear stunted and whose lower leaves are yellow. On light soils the effect is more striking than on heavy soils as the fungus interferes with the water flow and there is likely to be a water deficiency on light soils. Wet soils may encourage more of the disease but it will be later appearing. The dark wood ring is the most positive evidence.

The organism. *Fusarium bulbigenum* Cooke and Massee var. *lycopersici* (Brushi) Wollenw. and Reink. As indicated by the name, it was formerly known as *Fusarium lycopersici* Sacc., but Wollenweber and Reinking consider it to be a variety of *F. bulbigenum.*

The fungus may live in the soil for as much as ten years but it usually dies out in the first year. It does not form zones of growth on media and the macroconidia are often in pionnotes or pseudopionnotes. They are mostly 3-septate and from 30 to 40 by 3.3 to 3.5 microns.

Control. Rotation and sanitation. In New South Wales (Ag. Gaz. N. S. W. I. pp. 367-371, 1949) clean seed is the only recommendation. Bohn and Tucker (73) found no varieties of *Lycopersicum esculentum* Mill. either cultivated or wild that showed any resistance to the fungus. Plants of *L. pimpinellifolium* Mill. from Peru, South America, appear to offer hope as a possible immune plant for hybridization. Hybrids between this plant and susceptible commercial varieties are immune so that where the disease is severe this may be a solution to the problem.

Tomato Soft Rot

Host. Tomato.

Geographic distribution. Reported at Lyallpur by Madhok and Fazal-ud-Din (411).

Appearance on the host plant. The above authors report an under-skin rot accompanied by a discolouration, which turns brown and becomes wrinkled. If badly rotted the fruit becomes shrivelled and pulpy.

The organism. The organism, which they did not identify, was a round-ended rod measuring some 0.75—1 × 1.25—2 microns. It is motile with peritrichous flagella. The spores are oval, central and measure .75 × 1.2 microns.

Damping-Off Disease

Host plants. This disease is one of the most widespread known and has been reported on seedlings of practically every known plant. It is especially bad among nursery seedlings of all kinds.

Geographic distribution. World-wide.

Appearance on the host plant. The common name "damping off" describes the disease as well as any could. The most common symptom of the fungus is the sudden

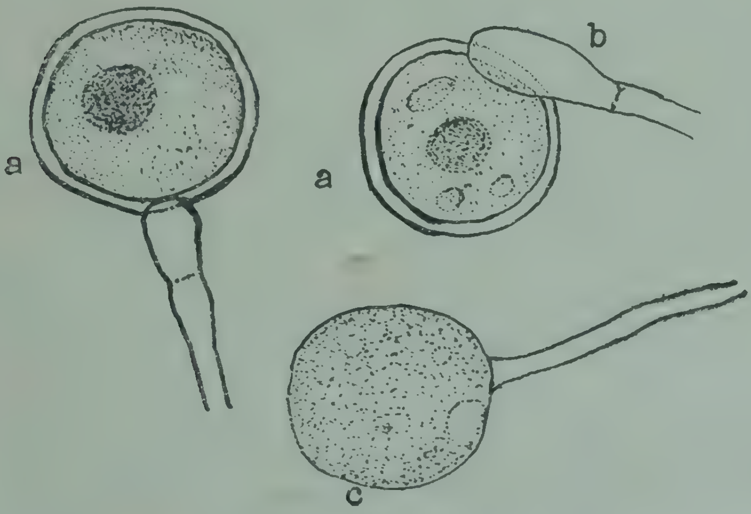


Diagram of the reproductive organs of *Pythium debaryanum.*

A. Oogonia.
B. Antheridia.
C. Sporangium.

collapse and falling over of the otherwise normal looking seedling. Upon examination the plant will be seen to be decayed at the ground line. The tissues are soft and watery in appearance and often give off a disagreeable odour. A lens may disclose a thin, cottony mycelium over the tissues.

The organism. The organism, which was first described as the "damping off" fungus, was described by Hesse about 1874 and called *Pythium de Baryanum* by Hesse. The fungus is delicate and likely to be variable in diameter. As the branches arise from the main hyphae they are smaller at first. The protoplasm is densely granular and there are numerous nuclei scattered throughout. At first the hyphae are apparently intracellular but later they become intercellular.

The conidia are intercalary or terminal and are thin-walled, nearly round bodies some three to five times the diameter of the hypha from which they arise. Germination is by a short tube through which the contents extrude to form a thin-walled vesicle within which a number of swarm spores (zoospores) are formed and these escape with the rupturing of the vesicle. The zoospores are biciliate with lateral cilia. Some of the conidia are decidous and germinate immediately by forming zoospores without the vesicle.

Oogonia are formed as terminal or intercalary bodies and are of much the same form as the sporangia. They are cut off from the vegetative cells when two or three times the size of the hypha. The oogonium is divided into two regions of density; the outer, or periplasm and the inner or ooplasm. The inner portion is filled with dense cytoplasm and many nuclei. As the oogonium matures the nuclei, except one, migrate to the outer portion and become the outer portion of the ooplasm and the central portion becomes the egg with a single nucleus.

While the oogonium is developing, on another branch of the same, or neighbouring hypha, there is developing a smaller body containing dense cytoplasm and a single nucleus, which functions as the male gamete, bearing a body known as the antheridium. This body is attracted to the oogonium and as the two come into contact a conjugation tube is produced from the antheridium into the oogonium and the contents of the male organ pass over into the egg. Fertilization takes place with the union of the male and female nuclei and soon a thick wall is laid down around the resulting oospore.This usually measures from 15-18 microns depending upon the conditions. The zoosporangia are somewhat larger.

Life cycle. The fungus may live either parasitically or saprophytically in the soil and thus the question of elimination becomes a serious problem. Where the means are available, soil for nursery beds may be steam-sterilized and the saprophytic condition is thus eliminated but this is not possible for the average farmer so that he has to depend upon other measures for control. The fact that the fungus may live on many weed hosts means that about all that is needed for an epidemic of damping off in the nursery is to produce the optimum conditions for the fungus. At the same time there is no doubt that the oospores are also a factor in carrying the fungus over dry or cold conditions. They may remain in the soil for a number of months and then germinate upon the return of favourable conditions.

Thus a life cycle may be described by beginning with the oospore germination and the production of swarm spores. These find the tender stem of a seedling and immediately settle down, lose their cilia and produce a germ tube which penetrates the soft tissues and then by rapid growth the entire stem is invaded with the resulting damping off and collapsing of the plant. If the plants are close together the fungus may spread from one to another and thus in a short time many of the plants may die. As the tissues become decayed and the fungus is growing rapidly the sporangia and zoospores are produced in large numbers. As the food becomes scarce, the oogonia are produced and after fertilization the oospores are there for the dry or cold period.

Control. Of course the most important method of control for the farmer is clean seed beds where that is possible. But in many cases this is impossible and therefore other methods may have to be used. Good drainage and planting so that there will be good air drainage is one of the best methods of control. The fungus is one that thrives best under very humid conditions; therefore reducing the humidity will retard the fungus.

In some cases formaldehyde, one half fluid ounce to one gallon of water to each foot of soil, applied the day before planting and covered with paper, will prevent the appearance of the fungus. Sometimes copper sulphate and zinc chloride are applied. As the fungus cannot attack plants after they become woody stemmed, it is only for a short time that the protection is needed.

Leaf Mould of Tomato

A minor disease of the tomato is leaf mould caused by a species of *Cladosporium*, one of the fungi with dark webby mycelium.

Hosts. The tomato.

Geographic distribution. Europe, American and the Orient.

Appearance on the host plant. The first symptoms are usually on the upper surface of the leaf. The first spots are diffuse white spots which rapidly enlarge and become yellow. When the weather is humid the surface becomes coated with a velvety olive brown layer of fungus hyphae. If optimum conditions prevail for any time the leaves may be killed and even the stems, fruits and blossoms may be infected. Spores are scattered by wind, water and any insect, bird or animal coming in contact with the leaves. Humidity of 90-100 is necessary for spore germination which is the reason for the heavy infection during humid weather. If the humidity is below 90 there is little likelihood of much disease. If spraying is done for any other disease it will also help control the leaf mould. Resistant varieties have been developed in the United States for greenhouse growing where the disease is severe. At present that does not seem necessary for India.

The organism. Cladosporium fulvum Cooke.

The hyphae are creeping over the surface of the leaf. The conidiophores are erect, branching at the top and look like soft wool if in mass. Conidia are rounded to ovate and two-celled as they mature.

Control. Sprays, dusts and good aeration are best controls. As mentioned above, the growers in the United States are looking toward resistant varieties.

The Common Diseases of Chillies

The red pepper or chillies (*Capsicum annum*) is another of the new world plants introduced into India and which has become as much at home as though it were indigenous. It was found in the Central American and West Indian regions by Columbus who took it back to Europe. By 1600 it had spread to the eastern portion of Asia and was on its way to become one of the important items of diet for millions of people.

It is subject to a number of diseases but only a a few are of real importance. Perhaps the most serious of all is the mosaic disease caused by the viruses. Ripe rot is another that may be serious under optimum conditions for the parasite. The *Cercospora* leaf spot is common but does not do serious damage except at irregular intervals.

Virus diseases of Chillies

The virus disease of chillies appears associated with that of the tomato. Most of the tomato viruses will also infect chillies, although the symptoms may not always be as marked as on tomatoes. Potato viruses apparently are not as much at home with the chillies as on other hosts. Tobacco viruses will go to chillies in a number of cases.

Hosts. It would appear that the chillies and tomatoes are the two most important hosts. That is among the cultivated crops. There are weed hosts that are important as means of carrying the virus over a dry period or the rotation period of the crop. Such wild plants as *Solanum nigrum*, *Physalis peruviana* (the cape gooseberry) *Zinnia* and others are wild hosts.

Geographic distribution. It is evident that they are world-wide wherever the chillies are grown.

Appearance on the host plant. The symptoms of virus disease on the chillies does not always appear as on the host from which the virus came. The most common symptom seen in the gardens at Allahabad is one that resembles the bunchy top of tomatoes. The tops of the chillies plants are more or less bunchy, the leaves are small and often twisted, the fruits, of any, are distorted and small, they are also poorly coloured, the branches are small and grow close together so that they appear bushy. The leaves are usually a lighter green or even yellow. Such plants are never as tall as the normal healthy ones. The chillies plants appear to stand the virus better than tomatoes on the average and sometimes they may produce a fair crop with mild symptoms of the disease. Other symptoms of virus diseases are the distinct yellow and green mottling of the larger leaves without the bunchiness of the tops and the dwarfing the whole plant.

The organism. The virus does not appear to have been named on the chillies but the symptoms, and the fact that the tomato carries the *Bushy stunt Disease* (*Lycopersicum Virus* 4) make it appear probably that it is the Bushy Stunt organism causing the disease on chillies.

Control. The control of virus diseases is one of the most difficult of problems that the farmer has to face. Chillies and tomatoes should not be planted near each other. That has become a rule in the United States of America where the two crops are grown commercially. Sprays and rotations have not been successful. The disease is carried by insects and the control method which is successful must control the insect vectors. Rogueing of diseased plants and selection for disease resistant stock is of value and it is on the latter that the main hope of the grower is centred.

Ripe Rot of Chillies

Host plants. Cultivated species of *Capsicum.*

Geographic distribution. World-wide.

Appearance on the host plant. The disease appears on the host plant after the fruit has begun to ripen. The spots are usually sunken and darkened becoming covered with pinkish or dark fructifications which are arranged more or less in concentric circles. These are just visible to the naked eye or with a hand lens. The symptoms may be confused with anthracnose (*Colletotrichum nigrum* Ell. & Halst.) except for the lack of setae.

The organism. The name *Glomerella piperita* was given by Butler (93) in 1918, but appears as *Glomerella cingulata* (Stonem) S. & v. S. in a later work (96). The imperfect stage is known as *Gleosporium piperatum* E. & E. There is much confusion regarding the fungus. It has been described under a number of names. See Stevens (741) who gives a list of 14 different species of *Glomerella*, *Colletotrichum*, *Gloeosporium*, *Laestadia* and *Neozimmermannia* which it has been called at different times. He also states that when all of them are considered, there are some 34 different hosts for the fungus. If there are differences at all it would appear that these must be biologic forms.

The fungus forms a stromatic layer under the epidermis which produces the fruiting structure (acervulus) and from which the conidia and conidiophores arise. The conidiophores are densely packed together and the conidia are held together by a mucilaginous material and when seen in a fresh mount appear in long chains or rows of spores that soon separate in water. In mass they are pale pink in colour but individually they are hyaline, straight or slightly curved with rounded ends. They are some 11 to 24 by 4 to 5.5 microns. According to Stevens (741), the perfect stage has been found on media after about one month. The perithecia are dark brown pyriform, thinly membranous and hairy. The asci are club-shaped and the spores are slightly curved.

Control. Sanitation should not be difficult. Destruction of the diseased fruits and rotation is important.

Other Species of Colletotrichum on Chillies

Thomas (798) in 1941 reported on a comparative study of three species of *Colletotrichum* and concluded that they were identical. He studied *C. indicum*, *C. curcumae* and *C. capsici* and concluded that they should all be called *C. capsici* as that was the older name.

Cercospora Leaf Spot of Chillies

Hosts. *Capsicum annum.*

Geographic distribution. Where the pepper is grown.

Appearance on the host plant. The infected areas are small, ranging from 2—10 mm. or, where they merge, they may be larger. The margins are distinct and sometimes darker or even tinged with red. The centres are likely to be ashy gray as the conidiophores and conidia form. Infection is mostly on the leaves.

although petioles and young stems may also be infected. If young seedlings are attacked in the seed beds even damping off may occur.

The organism. Cercospora capsici Heald and Wolf.

Conidiophores are short and produced in clusters. They vary 30—50×6—10 microns. They are dark with a slight olive greenish tinge. Conidia are lighter in color, frequently tapering towards both ends. They vary from 20—60×5—8 microns. Septations vary from 3-8.

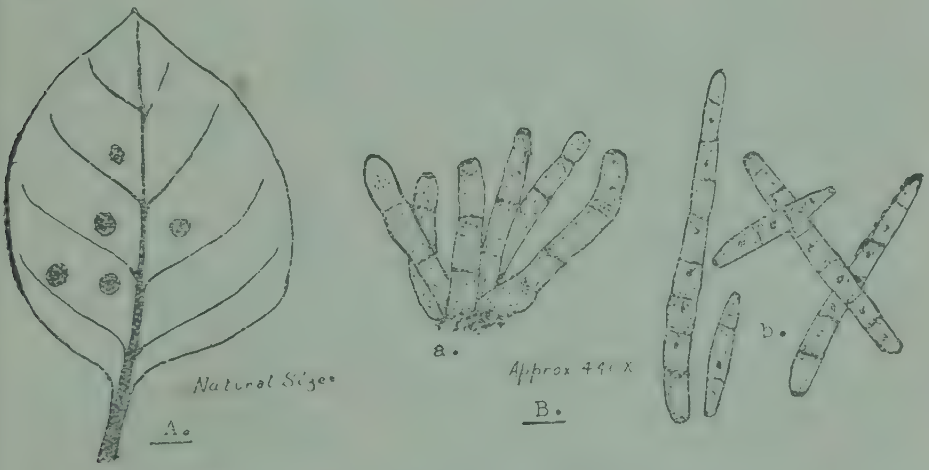


Diagram of a leaf of chillies illustrating the leaf spot caused by *Cercospora capsici.*

A. Leaf of chillies with infection spots.
B. a. Conidiophores. b. Conidia.

Control. Usually not serious and thus does not become a serious problem. Destruction of diseased leaves and wider spacing of the plants will aid in control.

THE COMMON DISEASES OF THE EGG PLANT
(*Solanum melongena*)

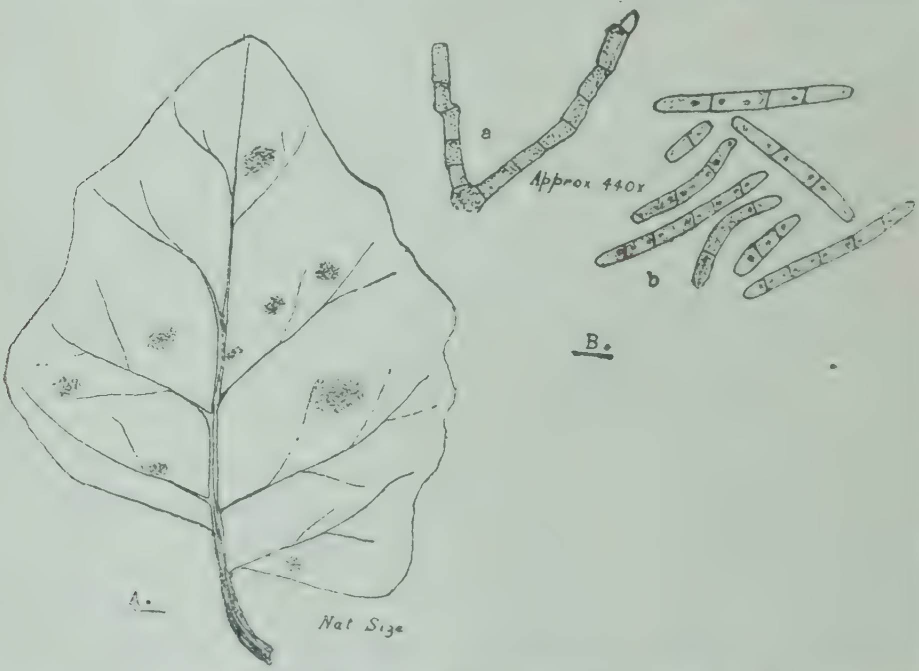
There are only a few diseases of importance on the eggplant. It is not seriously infected with virus diseases and it is not attacked by the early and late blight organisms as the potato and tomato. Perhaps the most commonly met with disease in the United Provinces is the *Cercospora* leaf spot. Seedling blight may be serious in the nursery before planting into the field. Root rot is serious under some conditions. *Phoma* canker may cause some loss.

Cercospora Leaf Spot of Brinjal

Hosts. Members of the *Solanaceae.*

Geographic distribution. General over the world.

Appearance on the host plant. The infections appear on either surface of the leaves as grayish, irregularly circular areas. At first the margins are not clearly defined but as the tissues become dried in the centre of the spots the margins become more sharply outlined.



Brinjal leaf diagramed to illustrate *Cercospora* leaf spot infections.

A. Diagram of the leaf.
B. a. Conidiophores. b. Conidia.

The spots become darker with age. As the leaves age they become yellowish and, if badly infected, they fall.

The organism. Cercospora solanacea Sacc.

The conidiophores are short, frequently septate light olive in colour, with distinct conidial scars. They measure 30—50×5—7 microns. The conidia are straight or slightly curved, septate with from 1 to 5 septations. They measure 10—40×4—5.5 microns.

Control. Destruction of the old infected leaf material and rotation so that no member of the genus *Solanum* follows in the rotation.

Blight of Egg Plant

Host Plant. Solanum melongena L.

Geographic Distribution. America, Europe and India.

Appearance on the host plant. The first appearance is a damping off of the seedlings which are attached some two inches above the soil line. A second symptom is the appearance of brown leaf spots from 2 mm. to 2-3 cm. in diameter, which are varied in shape from round to irregular. Later there are invasions of the fruit as well. The fruit infections result in circular, depressed, discoloured areas which are rotted.

The organism. Phomopsis vexans (Sacc. & Syd.) Hart.

The pycnidia appear on the dead tissue which shrivels and becomes darker. The conidia (pycnospores) are of two types. The *Phoma* type are single-cellet, 5-8 by 1-2.8 microns with two oil dropletts and the filiform curved type stylospores are 13-28 microns.

Control. Sanitation and crop rotation are probably the most practical control measures.

Root Rot of Brinjal

Hosts. The various organisms which appear associated with the root of brinjal have a wide range of host plants. At least all of the *Solanaceae.*

Geographic distribution. They are widely distributed.

Appearance on the host plant. In the field the many symptoms are more or less mixed so that it is impossible to say that this is due to one entirely. The first symptom noticed is a yellowing of the leaves. Flowers abort and the general appearance will be sickly.

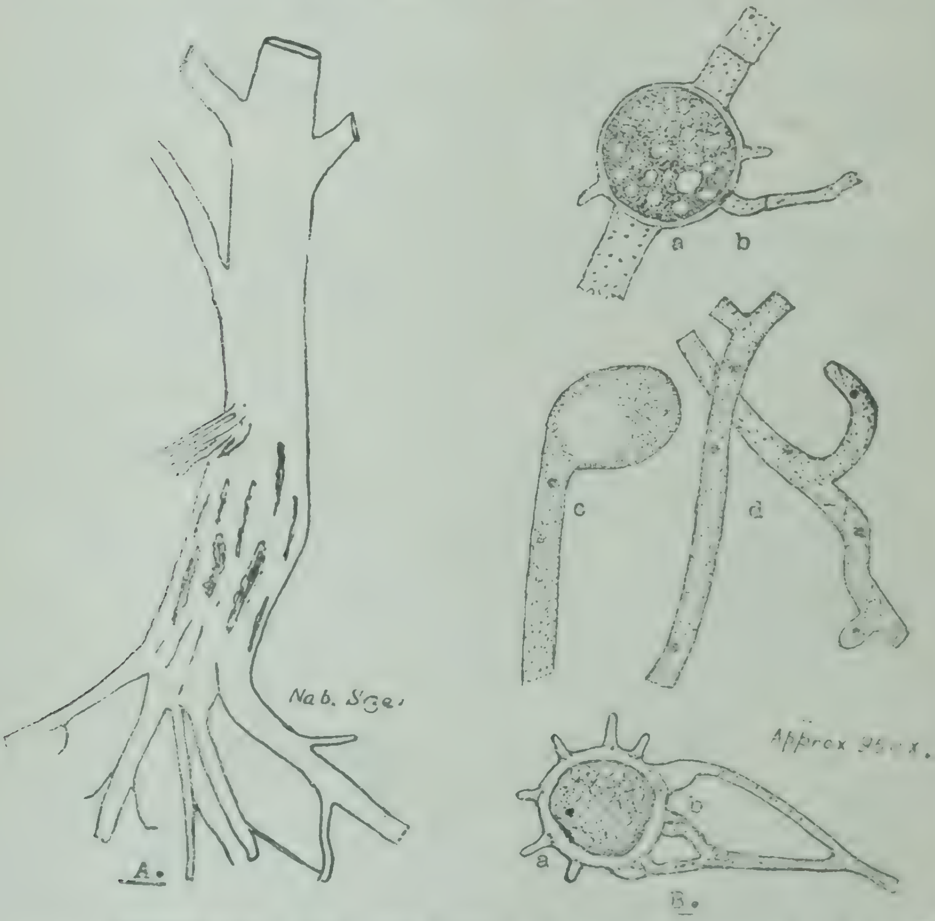


Diagram of a stem and portion of a root system of a brinjal plant, found on the Agricultural Institute Farm, Allahabad, from which a *Pythium* was isolated. In addition to the Pythium a species of *Rhizoctonia* and one of *Fusarium* were also isolated.

A. Diagram of the diseased roots and stem.

B. Diagram of the isolated *Pythium* fungus. a. Oogonia. b. Antheridia. c. Sporangium. d. Portion of mycelium.

As the rotting progresses the plant will appear loose in the soil and if bent will likely break at the surface of the soil. Examination of the roots at that time will disclose most of them blackened and decayed. From the margin between the decayed and the healthy tissue several fungi can be isolated that are known pathogenic. Among the most common will be species of *Rhizoctonia*, *Fusarium*, *Sclerotium* and others.

The organisms. Rhizoctonia solani Kuhn. This has been found commonly at Allahabad on decayed brinjal roots.

Fusarium spp. These have not been identified.

Sclerotium rolfsii Sacc. See under groundnut.

Pythium sp. One species of pythum, which resembles *P. mamillatum* Meurs has been found in the diseased tissues. See illustration.

Control. The general practice of rotation will help but destruction of the old diseased stalks will also aid. Organic fertilizers will probably do more for this sort of combination than any other treatment. The identity of the fungi is of importance from the mycological standpoint but as there are some fungi among them that organic manures have partially controlled it is not so important for the farmer that he know all the names to apply manure.

Phoma Canker of Brinjal Stem

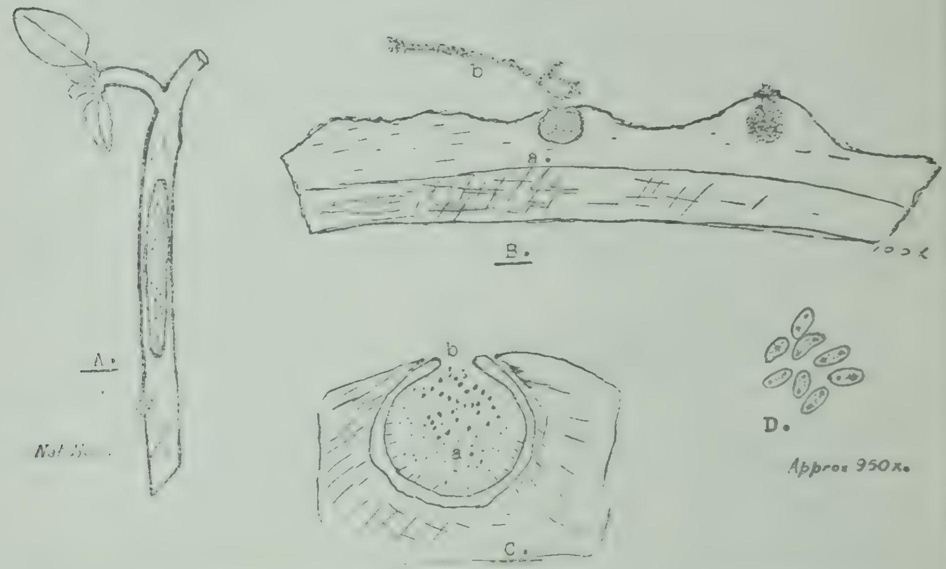
Hosts. Principally on brinjal.

Geographic distribution. Apparently world-wide.

Appearance on the host plant. It attacks the seedlings of the brinjal causing a damping off. On the older plants it causes a canker to form on stems and petioles. Inspection of these will show minute pycnidia.

The organism. Phoma solani Hals.

The pycnidia are small and imbedded within the tissue. They are flattened or rounded. The Pycnidia are oblong, hyaline, measuring 5—6×2—2.5 microns. They are ejected in a typical cirrus as shown in the diagram B, b.



Diagrams illustrating stem canker, caused by *Phoma solani*, on brinjal stems.

A. Diagram of a portion of a brinjal stem showing a canker.

B. Diagramatic drawing showing the pycnidia in the surface of the canker. a. Host tissue. b. Cirrus of pycnospores issuing from the ostiole of the pycnidium.

C. Diagram of a single pycnidium. a. Pycnospores. b. Ostiole.

D. Pycnospores enlarged.

Control. Rotation and destruction of old diseased plants.

THE COMMON DISEASES OF TOBACCO

Tobacco is grown under a very wide range of conditions which run from the tropical to the temperate. For that reason it has a large number of parasites which attack it. It is cultivated in every province of India including Assam. From the standpoint of amount of tobacco produced India is second to the United States of America only. One of the most common diseases of tobacco is the powdery mildew, *Erysiphe cichoracearum* D. C. which has been described under the diseases of *Cucurbitaceae*. It will not be discussed in detail here. Another common disease is the leaf spot caused by *Cercospora nicotianae* Ell. & Ev. Bacterial Wilt is also serious disease in some sections. It has been reported as common in Mysore. Other diseases are mosaic, stem rot, black shank, and root rots.

Tobacco Mosaic

There are at least 20 virus diseases which have been identified on *Nicotiana*. *Nicotiana Virus* 1, which Holmes (288) calls *Marmor tabaci* var. *vulgare*, will be discussed at this time. This same virus also causes the ordinary, or mild, mosaic of tomato, hence a separate discussion of the disease on that plant will not be given.

Host plants. Although this virus does not have the host range of *Nicotiana Virus* 12, it is probably of equal importance because of its relation to the tomato. Among the plants infected are species of *Ranunculaceae*, *Polygonaceae*, *Chenopodiaceae*, *Leguminosae*, *Compositae*, *Polmoniaceae*, *Hydrophyllaceae*, *Boraginaceae* and *Solanaceae*. A total of 9 families, 24 genera and 43 species of plants includes not all of the hosts but the most important.

Geographic distribution. Appears world-wide. Desai (180), Pal and Tandon (589) Uppal (845) have reported on virus diseases on the tobacco and tomato in India.

Appearance on the host plant. On the younger leaves of the tobacco plants the veins may show a clearing and later this may be followed by mottling. The leaves may be very much narrower than normal. Under high temperatures, the disease is much more severe than when the weather is cool. At such times the leaves may be chlorotic and deformed. On the other hand if the temperature goes too high, above 98 to 100 F., the symptoms may be masked. This is also true if the temperature goes below 50 F. Partial sterility may result from the virus. Smith (717) states that as much as 50% of the pollen may be sterile.

On the tomato, the virus causes a mottling with raised dark green areas. When temperatures go high mottling is severe but the stunting is less severe; on the other hand when the temperature is low, the mottling may be less but the stunting more severe. It may result in the production of the fern leaf type of leaves. In the Allahabad district the stunting and fernleaf are common but there is little of the anthocyanin formation

The organism. Nicotiana Virus 1.

The virus shows a positive reaction to the serological precipitin test. It is inactivated after one minute at 96°C. and three minutes at 93 C. It is easily transmitted by mechanical means. Even broken trichome [Smith (717)] is said to be sufficient to produce transmission.

Control. In spite of the ease with which the virus may be transmitted mechanically, insect vectors are not thought to be of much importance in transmission. The virus is resistant to heat and drying and this makes extreme care necessary when working about the plants. Jones and Burnett (324) state that tobacco mosaic was transmitted to tomatoes by workers using tobacco in the greenhouses.

Desai (180) believes that a bacterium is associated with the tomato virus. If this is proved it will mean that we shall need to widen our control measure applications to include bacteria as well.

Tobacco Leaf Curl

Pal and Tandon (589) studied a virus disease of tobacco in the Punjab which they stated was composed of some five types of symptoms. These they considered as types A, B. C. D. and X. They found leaf curl present to the extent of 5-10 per cent normally but it was epiphytotic in 1934-35. The general symptoms were a stunting, reduction of leaf area, a curling of the leaves, a thickening and greening of the veins. The inflorescence is shortened and the veins of the petals and ovary wall become thickened.

The authors report an interesting curve in the infection pattern for the season. Using a variety known as P. H-142 they found the maximum spread of infection from October to December with the disease appearing to be dormant from the first of January to March when it again appeared.

In 1935-36 plants from seed sown in June were 63 per cent more severely infected than seed sown in August which is the normal time. Leaf curl is rare in the seed beds but occurs after transplanting.

Differences between the types of viruses on the basis of symptoms was not clear. Some differences, however, did appear. A. produced small, curled, thickened leaves which were brittle and dark green. B. on the other hand produced larger leaves, less curled and thickened a pale yellowish green and no thickening or brittleness. C. and D. do not produce stunting. Each produce vein clearing but that of C is more intense than for D.

They found that A and B could be readily separated from C and D by grafting.

They were not sure of other hosts but found similar symptoms on several other plants, including *Zinnia elegans*, *Althea rosea*, *Hibiscus rosa sinensis*, *Crotalaria juncea* and *Scoparis dulcis*.

After some considerable work being done with the insect vectors it was established by Pruthi that the white fly (*Bemisia gossypiperda*) can transmit the virus, or viruses, from sunn hemp to tobacco.

Ring Spot of Tobacco

In 1938 Thomas (793) reported ring spot, along with mosaic and leaf curl, on tobacco in the Coimbatore district. Ring spot is also known as Nicotiana Virus 12 and produces as the first symptoms several single necrotic rings which become browned in colour in a few days. Succeeding rings may appear in four days and again in about five days. This may continue until there are a number of these necrotic rings alternating with normal tissue. The centre of the area dies within a few days and it then appears as though the centre was a very small ring.

Cercospora Leaf Spot of Tobacco

Hosts. Tobacco appears the main host but no doubt closely related weeds are also hosts.

Geographic distribution. Throughout India and Burma and the United States. Probably world-wide.

Appearance on the host plant. The disease was formerly referred to as frog-eye of tobacco. This was due to the narrow red border around the diseased portion. The centre of the spots are gray to white. The spots appear in any place on the leaf and, on young plants, may occur on the stems. The spots do not injure the ordinary tobacco but in the case of the cigarette tobacco it is a serious matter for the spots spoil the leaves for cigarettes. The spots may coalesce into larger spots thus spoiling the leaves for any use. As the diseased areas grow older they dry in the centre and that portion falls out leaving a ragged hole. The disease is seasonal and the climatic relations are not clearly understood. High humidity is very definitely associated with the disease. One difficulty with the disease is that spots may be so young as to appear inconspicuous to the examiner and yet under flue curing they may become very prominent.

The organism. Cercospora nicotianae E. and E.

The conidiophores are produced in tufts which emerge from the stomata. They are septate and show two to three conidial scars towards the upper end. Occasionally branched. They are septate and measure 75—100×4—5 microns. Brown in colour.

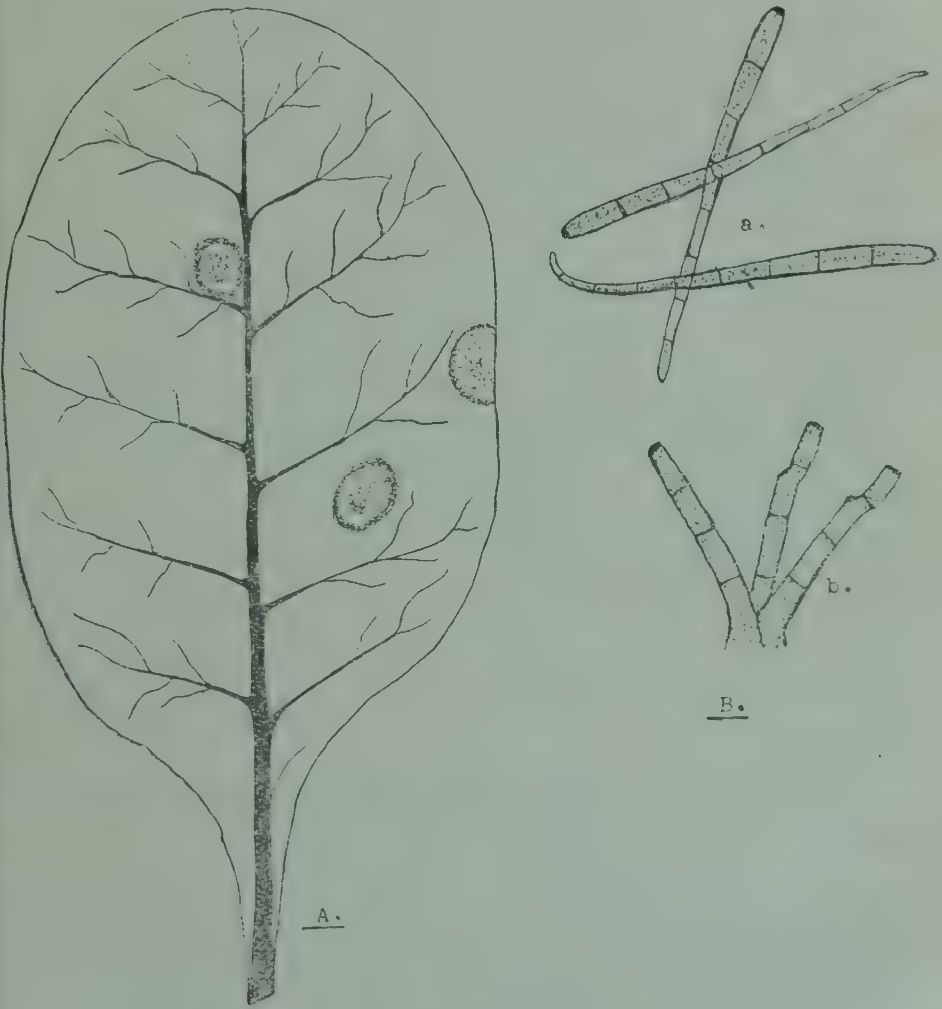


Diagram illustrating *Cercospora* leaf spot of tobacco.

A. Diagram of an infected leaf showing the typical spotting.

B. a. Conidia. b. Conidiophores.

The conidia are slightly curved, septate, hyaline and 40—50×3—3.5 microns.

Control. In many places the disease is of little importance but in some it is serious. Aiyer (9) states that if the plants are sprayed, at the time of topping, with a colloidal copper preparation some control may be had. Spraying results in increase of the crop and reduction in spot of flue cured tobacco.

Stem Rot of Tobacco

Host. Tobacco, *Hybiscus, Linum.*

Geographic distribution. World-wide.

Appearance on the host plant. According to Kheswala (331), the first symptoms are lesions on the stem at the ground level. The stem becomes lighter brown in colour at the place of infection and assumes a water-soaked appearance. The lesions being located at the ground level indicate a soil-borne organism. Infected stems usually bend over and at the fold of the bend a white mycelial growth takes place. Inside the stem thick ashy-brown sclerotial threads form and in the older stems elongated sclerotia may be found. The entire pith region may be filled with mycelium. The middle lamella will be destroyed and the tissues much disintegrated.

The infection takes place through the roots, by way of wounds or by ascospores.

The organism. Sclerotium sclerotiorum (Lib.) DeBary. The mycelium is first white and becomes brown later. The threads thicken and form the elongated sclerotia referred to above. Sclerotia range from 4-15 mm. when grown on oatmeal agar. Apothecia grow best at low temperatures, the number and size depending on the size of the sclerotium but varying from 3-15. The asci are cylindrical, typically 8-spored. They measure 81—127×6.8—10.2 microns, the mean being 110.25×.45×8.6. Paraphyses are long and filamentous.

The optimum temperature is between 20 and 25°C.

Control. Rotation. Shallow ploughing and permitting the sun to strike the exposed soil bearing the sclerotia. High temperatures kill them.

Bacterial Wilt of Tobacco

The bacterial wilt of tobacco (*Bacterium solanacearum* Smith) has been in India for some time. However, it does not seem to have become widely spread until recently. It has been in the district of Bengal for at least 30 years and may have been in other parts but not reported. Mitra (Int. Bull. Pl. Prot. XI pp. 85-87, 1937) reported in the Punjab in 1936.

Hosts. It has an extremely long host range. Over 100 plants scattered through some 24 families which included many of the common plants of India such as the banana, the canna, the groundnut, the bean, the pea, cotton, tobacco, tomato, cape gooseberry, potato, egg plant and many of the flowers and weeds of the field.

Geographic distribution. It is known over most of the world.

Appearance on the host plant. It is a vascular disease that causes wilting, dwarfing, and a brown stain of the vascular bundles. The base of the stem and the main roots are discoloured and partially rotted. If the stem is split open it is seen to be discoloured from the surface to the pith. In tobacco and potato the veins sometimes are browned. The destruction of the pith is such as to leave cavities filled with bacteria. In some cases the tissues shrink at the point of infection.

The organism. Bacterium solanacearum Smith. There are many synonyms for the organism such as; *Bacillus nicotianae* Uyeda, *Bacillus musae* Rorer, *Pseudo-*

monas solanacearum Smith, *Phytomonas solanaceara* (E. F. Smith) Bergey et. al.

The organism is a rod-shaped bacterium, motile by one polar flagellum, possesses no spores, no capsules. It measures 0.5 × 1.5 microns. It is gram negative, aerobic, produces no gas or acid. Its temperature range is from 10 to 40 with an optimum of 35-37°C. It has a thermal death point of 52°C.

Control. Seed bed sterilization with steam, where possible, disinfect the seeds with mercuric chloride and spray the young plants with Bordeaux mixture.

Butler suggested that summer ploughing and allowing the soil to remain open to the sun would destroy the organisms as they have a thermal death point of about 50°C. The soil often reaches that temperature in the hot days of May and June.

Entrance to the plant is probably made possible through wounds. Insects may be vectors but if so it has not been shown which ones.

Other Diseases of Tobacco in India

Black Shank of Tobacco

The disease known as "black shank" has been in India for some time. There is not much about it in the Indian literature but it has been found repeatedly in wide range of host plants in other countries.

Hosts. Tobacco, *Citrus*, castor, sesame and a variety of other plants.

Appearance on the host plant. As the name indicates, on tobacco it causes the stem to turn black at the surface of the ground and a short distance above. As the plants grow older the leaves yellow and then wilt. In the early stages it may cause a damping off of seedlings. Microscopical examination will not show any mycelium, unless under exceptional cases, but the tissues will be decayed and darkened. The decay may run down into the roots in some cases.

The organism. Phytophthora parasitica Dastur var. *nicotianae.*

Mycelium white, sparingly branched and non-septate. Sporangia are sparce, prominently papillate and broadly ovate. They measure 25×30 microns. The reproductive organs are typical of the *Phycomycetes* with the antheridia persistent. Tucker states (823) that there are two strains; one with large oospores with a mean diameter of over 20 microns and another with the mean diameter under 20 microns.

Control. Sundararaman (766) stated that control was obtained by burning trash on the seed beds, by treating the seed with 1 : 1000 bichloride of mercury or spraying the young plants with Bordeaux mixture. Galloway (238) reported a *Phytophthora,* which he considered *P. parasitica,* on tobacco in the Madras area which, when taken to Pusa, was active from April to October but apparently dormant during the rest of the year. That made the problem of control there somewhat less of a problem. In Nagpur a species of *Phytophthora* was found on tobacco seedlings in 1935 but Bordeaux 4-4-50 and removal of the infected plants controlled the disease.

Pythium on Tobacco

Pythium species have been reported on tobacco from time to time. Venkatarayan (892) reported *Pythium aphanidermatum* on tobacco seedlings which was controlled by applying 1 per cent Bordeaux mixture.

In Bengal, *Pythium de Baryanum* was controlled by use of formalin at the rate of one fluid ounce to two gallons of water.

Other Fungi

Macrophomina phaseoli has been recorded on tobacco by Likhite in 1936 in Baroda.

Powdery mildew has been recorded on tobacco and was controlled by removing the lower leaves and spraying with Bordeaux. The powdery mildew in this case being *Erysiphe cichoracearum* DC which has already been described under *Cucurbit* diseases.

CHAPTER IX

THE DISEASES OF SUGAR CANE, RICE AND COTTON IN NORTHERN INDIA

THE COMMON DISEASES OF SUGAR CANE

Sugarcane is one of the most important crops of the tropical parts of the world where rainfall and soil conditions are sufficient to meet the requirements of the crop. It has been carried over the world and exchanges of plants has taken place between most of the countries where it is grown. As a result there has been an exchange of varieties and with them the diseases they were afflicted with in the sections where they originated. As a result there are many serious diseases of the cane crop in every country producing cane sugar.

Among the most important in most sections will be red rot caused by *Colletorichum falcatum*. Another common one in India is smut. Bacterial rot has been severe in some sections, Virus diseases are serious in some sections, *Macrophomina* root and stem rot, collar rot, *Curvularia* leaf spot, Cytospora disease and a number of minor diseases have been reported and are present on cane in the United Provinces.

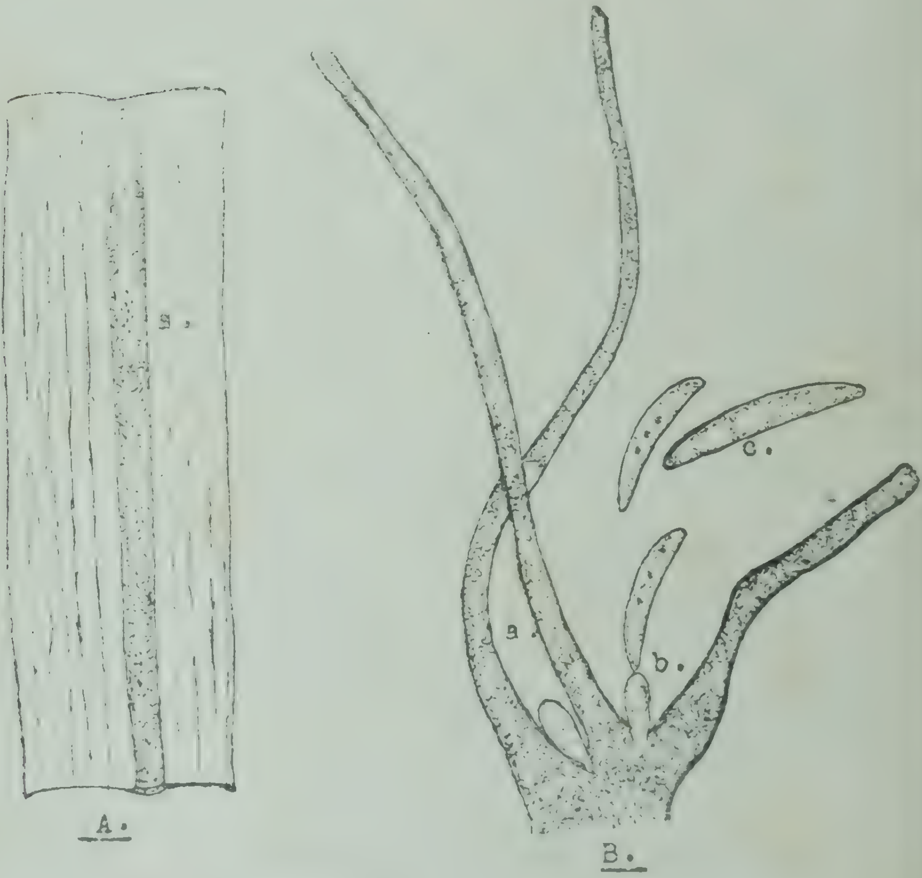
The Red Rot of Sugar Cane

Host plants. Species of *Saccharum.*

Geographic distribution. World-wide.

Appearance on the host plant. The common name 'red rot' describes one symptom of the disease. Perhaps the name adds to the significance of the disease but it is without question the most serious limiting factor in sections where it finds optimum growing conditions. It has been known for at least 48 years in the cane growing regions of Asia having been reported in Java in 1893.

The first symptoms of the disease will be yellowing and drooping of the leaves. The margin and tips wither and later the entire top may wither. The canes themselves show little evidence of the disease



Diagrams illustrating red rot on sugar-cane leaf.

A. Portion of leaf with infected area along the midrib. a. Infected area showing acervuli.

B. a. Setae. b. conidium and conidiophore c. conidia.

until a severe stage has been reached. Then there is a tendency to shrivel with a wrinkling of the rind.

When such a cane is split lengthwise and examined it will be found to contain red blotches throughout the length of the pith, some of which will be of several inches in length separated by bands of white extending across the stem. As the spots grow older the red gives place to a brown colour. If the piece of stem is placed in a moist chamber, soon a grayish white mycelium will grow out of and over the tissue and later small dark spots, acervuli will appear.

A common character is the breaking of the leaves in the middle, the broken half drying and the remaining portion retaining its green colour with a bright red patch at the point where the leaf broke. In this respect there is a similarity between the red rot of cane and the red spot of jowar caused by *Colletotrichum gramnicolum*. A sour odour is given off by the diseased tissue.

The organism. Colletotrichum falcatum Went. The mycelium is slender, much branched, colourless, septate and contains a distinct-type of oil droplet which is constant and can be used as an identification mark. It is found mostly in the spongy portion of the stem, rarely being able to enter the vascular bundles. The hyphae are both inter and intracellular. The red colouration is due to the action of the fungus. It is known that some cells will be more highly coloured than others. The vascular bundle cells are more strongly stained than the others. The staining is often some distance beyond where mycelium is.

In India today there are no commercial varieties of cane that may be considered ready for distribution as resistant to red rot. Some are showing less susceptibility to the red rot and mosaic than others. For example, Co. 290 is some what resistant to red rot in the North West Frontier but is being replaced by Co. 419, which is a popular variety in the Bombay area but will lodge badly on rich soils.

In the line of hybridization a number of different lines are being followed. Generic crosses involving *Saccharum*×*Sorghum*, Zeae×Sorghumm, and crosses involving bamboo have been made. These have so far proven of more interest than value but offer a possibility for future breeding. Interspecific crosses involving *Saccharum officinarum* with S. *spontaneum*, S. *robustum* and others some good results have been obtained. S. *spontaneum*×S. *robustum* has given some of the most popular varieties but there is still much to be achieved in the way of disease resistance.

In 1936 Serrano (Philip. Jour. Sci. LVIII, pp. 481-493, 1935) suggested the use of disease free setts, ratoon only disease free fields, use resistant varieties and plant crops other than sugar cane which are not to be exported from the farm or, preferably, from the field.

In 1928 Uppal (835) recommended the plowing of the fields before the hot season and thus exposing the fungus to the sun as it has been determined that it cannot live more than three months if exposed to the open sunlight.

Mathur (425) states that in the United Provinces *Colletotrichum falcatum* sporulates most readily during September and October. He observes that badly infected canes should be cut and the green portions fed to stock but the drier portions should be burned. Rogueing can also be done and isolated canes can be cut out. At Allahabad (1947) sporulation was abundant by the middle of September and continued to increase up to the end of September. This increase was no doubt due to the high humidity which persisted throughout the month. Mathur (425) states that only healthy setts be planted.

It does not sporulate readily in nature but acervuli may be found on the surface of the diseased leaves and stem. On the leaves they occur on the midrib in dark grayish areas. On the stems they are found in the neighbourhood of the nodes.

As in the case of *Gloeosporium*, the mycelium collects beneath the epidermis in stromatic masses from which the conidiophores are produced. As they elongate they rupture the epidermis and then produce conidia at the tips. Setae surround the acervuli. These are reddish-black, darker at the base and lighter at the tip, measuring from 100-200 microns and contain an average of 4 septa. The conidia are sickle shaped, usually have a single oil droplet in the center and measure from 20 to 30 by 5 to 7 microns.

Abbott (4) after comparing 85 isolations of the fungus from different sections of the world, concluded that there are two morphologically different groups based on the colour of the mycelium. One is dark gray and velvety and the other is nearly white and cottony. The light strain is more virulent on P. O. J. 213 in Louisiana than the dark strain. He considered them biologic forms.

In 1938 Abbott, (5) made a report of surveys of the cane growing regions of the U.S.A. and reported that the dark strain had predominated in the syrup producing states from 1930-1937 but that in 1938 the light strain predominated. He found that the light strain from P.O.J. 213 was more virulent than the light strain from other commercial varieties. He states that the differences in virulence were observed and that the five physiologic races were distinguishable from each other although the differences were not always well defined.

The question of transmission is somewhat debatable but a number of workers believe that borers are largely responsible for the spread of ret rot. Abbott (5) believes that borers cause the stem infections. Atkinson (37) reported that borers carried the fungus up and down the stems. On the other hand Padwick (571), reviewing the work of Abbott and others, says that Indian experience does not justify the statements regarding the part played by borers in the spread of red rot. He summarizes the sources of infection and the modes of entry into the plant as follows:

Sources of the fungus. (1) mycelium in the mother setts; (2) spores from diseased shoots; (3) spores from leaf spots; (4) spores from old decayed canes; (5) spores from mycelium in the soil and (6) spores from alternate hosts.

Modes of entry into canes (1) from mother setts to new shoots; (2) through borer holes; (3) through root primodia and leaf scars; (4) through the cut ends of setts and (5) through miscellaneous injuries.

Modes of entry into the leaves: (1) direct through the epidermis; (2) through leaf punctures made by insects; (3) through miscellaneous injuries. It would be well to read the article by Padwick in connection with the study of red rot.

It might be well to note here that Abbott (3) states that leaf infections furnish the conidia for the stem infections and that conidia from old stubble, trash and rhizomes furnish the inoculum for the leaves.

Carvajal and Edgerton (98) found the perfect stage of *Colletorichum falcatum* to be *Physalospora tucumanensis* Spez.

Control. Dey (185) states that red rot of sugarcane can be controlled by thorough sanitation and four or five year rotation. If this program is combined with the use of resistant varieties it shoud prove useful in controlling the disease.

Resistant varieties are the most hopeful control measures now being considered. Abbott, Summers and Rand (3) stated in 1936 that they had classified all of the canes at the Houma, Louisiana station into four classes on the basis of their reaction to the fungus as follows; (1) resistant; (2) moderately resistant; (3) susceptible and (4) very susceptible. At that time they had 214 varieties in classes 1 and 2 and 205 in class 3. The 22 varieties in class 4 they considered as dangerous commercially. This should furnish a basis for future classification and selection. Brandes and Sartoris (79) have written a good review of the sugarcane breeding work and their article should be read in this connection.

Sugar Cane Smut.

Host plants. It is found on varieties and species of *Saccharum.*

Geographic distribution. It has been reported in the Orient and in the sugar cane growing areas of Africa and Italy. In India it is found mostly in the cooler regions.

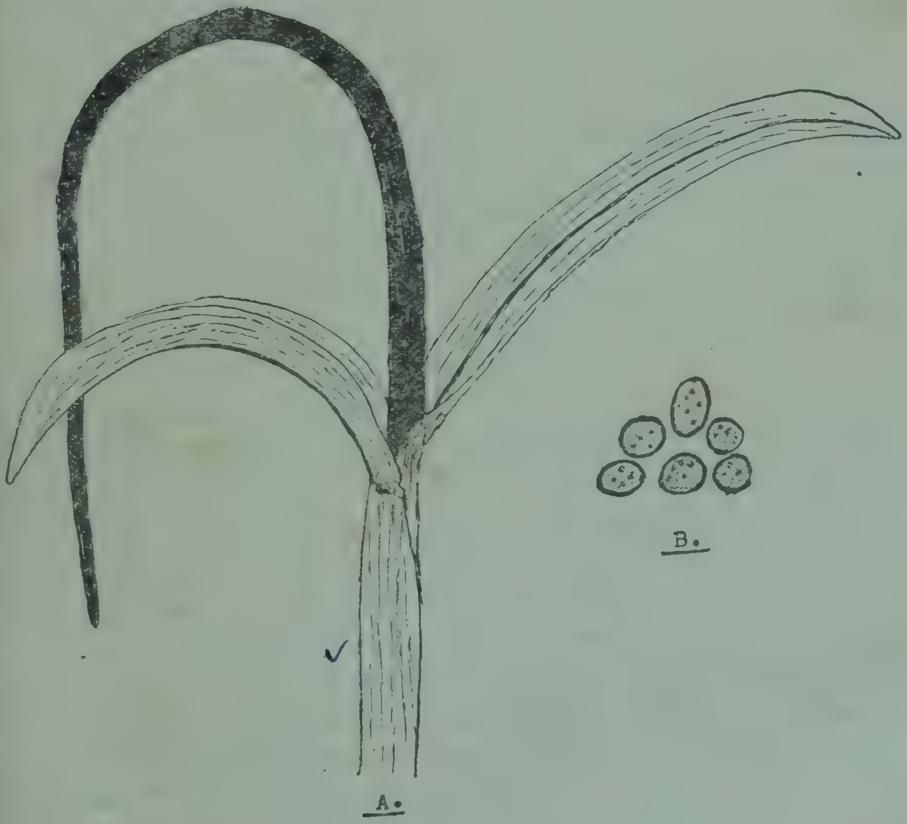


Diagram illustrating sugar-cane smut.

A. Smutted whip shown in black.

B. Spores. These are small and relatively thin walled.

Appearance on the host plant. The most characteristic symptom of the disease is the long whip like growth, dusty black in colour which protrudes from the top of the infected plant, often several feet in length. It has been thought that the floral shoot is the source of the whip but it is usually the non-flowering types that are the most susceptible to smut.

Secondary shoots may arise from the base of the stalks but not from the upper part. To the ordinary observer the only effect of the smut upon the plant is the production of the shoot.

The organism. Ustilago sacchari Rabenh.

It was named *U. scitaminea* by Sydow (96) and that name is used in the Philippines, East Indies and Java.

Only on the black whip will spores be found. They are spherical, smooth, brown and from 8 to 11 microns in diameter. Among them are numerous colourless thin walled sterile cells. Germination of the chlamydospores is typical of the smuts with the production of a septate promycelium and slender, straight sporidia on small sterigmata.

Lee (Sugar News XII, pp. 220-221. 1931) stated that transmission of the disease in the case of Uba cane was by cuttings from diseases plants. Where this was done the disease increased and it was evident that if the practice of using diseased plants for cuttings was followed long there would be serious consequences.

Control. Destruction of the diseased plants and the use of only healthy plants for cuttings.

Mathur (425) suggests that a careful examination of the setts before planting is necessary. Set aside a field for seed for succeeding seasons. Between April and June remove the smut whips and destroy them by boiling them. Trained boys may do this. Be sure that the smutted plants have been completely removed from the plots. Sundararaman (767) reported that it was completely removed from some of the districts by roguing. Francis (231) reported that roguing over the Madras area reduced the smut from 7 plants an acre in 1935-36 to 1 to an acre in 1937-38. The cost of the roguing was about 8 annas an acre. Padwick (571) stated that 1940 selections from Co.313 with 70-80 per cent infection produced only 3 per cent after roguing. Luthra and Sattar (406) state that smut is carried over from one plant to another by (a) planting setts from infected shoots, (b) spores borne in buds, (c) infection of the buds of standing canes, (d) ratooning from smutted plants. They found that germination of the canes from smutted plants was only 16 per cent as compared to 44 per cent from the healthy. They say that ratooning from the diseased plants was a serious source of infection. For that reason it was of especial importance that clean setts be used. Chona (110) states that there are two periods when the smut whips are forming. The first is in May and June and the second is October, November. Roguing must be done in both these periods. The May-June infections arise from the infected setts where as the October-November whips come from secondary infections.

Chona (110) reported that in some cases the infections formed galls on the leaves. Early infections caused the clumps to be spindly, or with small glossy shoots. Later infections may produce nearly normal stalks. Knowing these characters makes roguing possible.

In 1943 per cent of smut was on the increase. Co. 213 and Co. 299 were badly infected at Gorakhpur (110) Co. 312 showed 12-50 per cent infection at Fyzabad. Roguing was effective in the control of the smut. Destroy smutted plants. Burn them. Dipping the setts in water at 55°-60° C. for 10 minutes controlled the smut when 87 per cent developed in the controls (110).

In 1945 (424) the Imperial Council of Agricultural Research stated that when 108 varieties of sugar cane were dipped in a smut suspension 18 were fairly resistant. They report that the dipping in water at 55°-60° C killed the smut but reduced germination.

Mathur (424) stated that Co.312 and Co.313 are so susceptible to smut that they should be reduced in the planting and that Co.356, Co.419, Co.421, Co.395 and P. O. J. 2878 should be encouraged in the areas where smut is serious.

Sugar Cane Rot

Under this title will be discussed the bacterial plant pathogens which have been associated with the rot of sugar cane. There are at least two organisms, *Bacterium* (Er.) *sacchari* Ralden and *Bacterium pyocyaneus* Desai.

Hosts. The bacteria are not known on host plants other than sugar cane at this time.

Geographic distribution. Considering the present state of the nomenclature, it is difficult to determine the distribution. It seems from reports already received that it is likely to be found in most of the sugar cane regions of the orient.

Appearance on the host plant. Ralden (633) reported a stem rot of sugar cane had occurred in the Philippines which was characterized by a yellowing, or a yellow-browning, of the entire foliage which was followed by a wilting and rolling of the leaves. The inner tissues of the stalk soften and there is a rotting of the side buds. A disagreeable odour is given off and the whole portion appears as though scalded.

In 1935 Desai (181) reported the finding of an organism of the *Bacterium pyocyaneus* group causing a rotting of the cane stalks of Co. 300 and Co. 313 with an accompanying disagreeable odour. He found two strains of bacteria, one blue and the other white, associated with the disease. Neither of them appeared to cause the disease unless they were deeply imbedded in the tissue and then the blue strain caused a rapid decay. The white strain alone seemed unable to cause decay but associated with the blue strain was able to cause rapid decay. From the descriptions of the bacteria causing the soft rot of sugar cane in the Philippines by Ralden (633) and the one causing a similar disease in India by Desai (181) one might believe they were discussing the same organism.

The organism. Ralden (633) named the one isolated from cane stems in the Philippines, *Bacterium* (Er.) *sacchari.* He found it a short rod which grew in pairs, singly or in chains. Sometimes it was found in clumps. The measurements were 0.95-2.2× 0.5-0.7 microns. It stains readily with ordinary stains, is motile with four peritrichous flagella. It is this last character which distinguishes it from the closely related organism *B. pyocyaneus*, which is motile by from one to three polar flagella (see previous paragraph) and thus according to Ralden makes the separation of the two genera justified.

Control. The one found in India has not yet been positively identified. Up to the present time control recommendations have not been definitely formulated but certainly rotation of crops in the localities where the rot occurs, together with sanitation and destruction of the diseased plants, should do much to check its spread.

Virus Diseases of Sugar Cane

Fortunately for the Indian cane grower only two of the four serious sugar cane virus diseases are known here. Mosaic and streak are known but the Fiji Disease and Serah are not known although they have been reported in the Dutch East Indies. Dastur first reported mosaic in 1921 at Pusa. Streak was seen in the C. P. some time later and appears to have been introduced from Coimbatore. Motz stated that the Indian strain of sugar cane mosaic was identical with the highly infectious sugar cane virus 1 B, of Summers in Louisiana, U. S. A.

Sugar Cane Virus I

Host plants. The host range includes sugar cane, sorghum, pearl millet, sudan grass, wild sugar cane (*Saccharum narengo*) crab grass (*Syntherisma sanguinalis*), yellow foxtail (*Chaetochloa crusgali*) Panicum spp and goose grass (*Eleusine Indica*).

Geographic distribution. World wide in the cane growing regions. It is thought to have originated in the eastern part of the world.

Appearance on the host plant. The first symptoms are the pale patches, or blotches, on the leaves which are several shades lighter than the regular green colour. These areas are irregular in size and parallel to the long axis of the leaf. The symptoms are most distinct on the newly unrolled leaves in the case of some varieties. The virus may not produce any other symptom but in other cases the plants may be dwarfed, develop necrotic areas on the leaves and produce only a bunch of twisted leaves at the top. The general appearance of an infected plant is a mottling of the leaves produced by white blotches and streaks on the green back ground of the leaf.

The organism. *Saccharum virus* 1.
Sugar cane mosaic.
Yellow stripe disease of cane.
Sugar cane mottling disease virus.

The virus is killed at a temperature of 53-54 degrees C. Chona (111) stated that the mosaic virus in India varied as to the thermal death point. It was 45°, 55° or 60°C. according to source. Just why this was so was not clear but the thermal death point did not change regardless of the treatment.

The general effect of the virus is to reduce the yield of cane both as to tonnage of stalks and of juice percentage. However Chona (111) states that there is no loss of percentage of sugar in spite of the loss in cane weight i.e., the percentage of sugar per unit of cane remained the same as for the normal canes. On the other hand McRae (448) stated that mosaic on Co. 213 caused a loss of 8 per cent in germination, 14.8 per cent loss in the stripped cane, 8.9 per cent loss in juice and 4 per cent loss of sucrose. Sundararaman (607) stated that mosaic caused a loss of 10 per cent. In Patna the mosaic infected plots were reported to have lost some 19 per cent in yield as a result of mosaic.

Control. Control is largely a case of using disease free setts, roguing and using resistant varieties. In 1935 Luthra and Sattar (394) found that some varieties remained unaffected. They were Co. 214, Co. 227, Co. 232, Co. 233, Co. 238, Co. 243. They stated that roguing was effective only on the disease resistant varieties. Sundararaman (767) reported that Co. 326, Co. 229, Co. 335, Co. 355 Co. 356, Co. 508, Co. 413 and P. O. J. 2878 remained free from the disease in the trials. Thomas in 1939 (795) reported that Co. 434, Co. 511 and Uba A were unaffected by mosaic. In 1941, the same author (798) stated that Co. 205, Co. 422, Co. 434, Co. 508 Co. 311 and Uba remained free from infection that they were considered resistant. Chona (111) stated that Co. 214 remained free from the infection even after artificial inoculation. At Pusa, Co. 213, with 100 per cent infected canes did not lose more than 10-12 per cent in tonnage with no loss in per cent of sugar. The same was true of Co. 213 at Patna and at Cawnpore. The Surkha Saharanpuri variety suffered 18-20 per cent in cane but no loss in per cent of juice or in juice quality. The student will wonder why this is brought in but in foreign countries there has been the contention that mosaic caused a loss, not only in tonnage but in sucrose content and in quality. This would indicate the Indian strain is not as serious on sugar cane as those of other countries. However even here it is a serious problem for with some 4.000,000 acres in sugar cane a 10 per cent infection would produce a 1 per cent loss in yield and that would amount to some Rs. 3,300,000 per annum.

Reguing and planting only clean setts will control the disease.

Macrophomina Rot of Sugar Cane

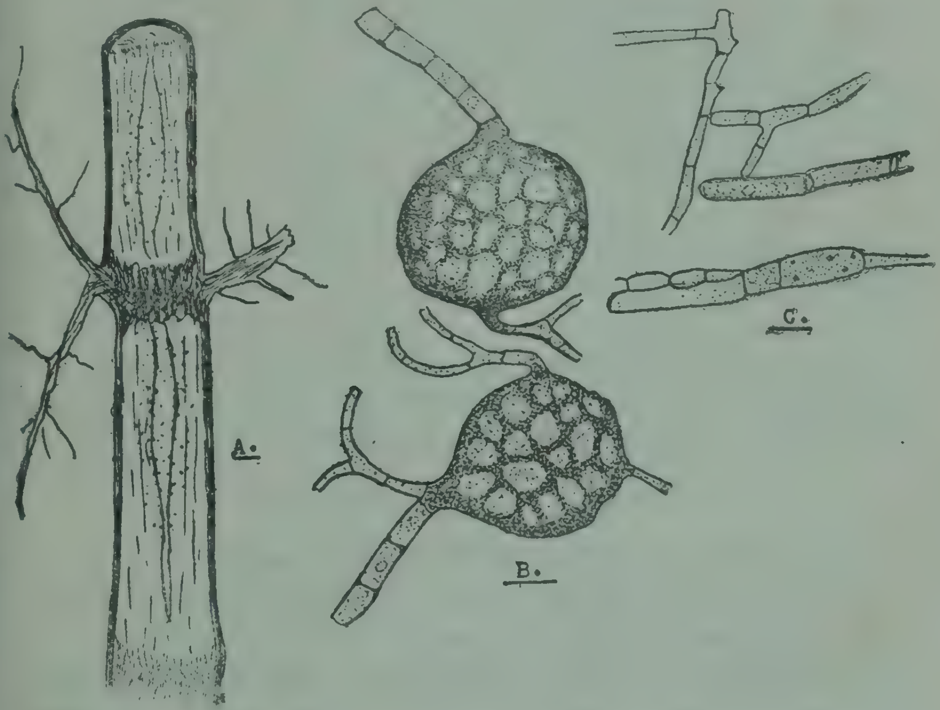
Hosts. Practically every major crop on the farm and many other plants.

Geographic distribution. World-wide.

Appearance on the host plant. The first appearance of the rot is a stunting and yellowing of the stalks infected. They will be seen to have shorter internodes and the terminal point does not mature. These characters are not sufficient to identify the disease from other root and stem disease but they will aid in locating the stalks which are infected. Infected stalks will appear somewhat lighter in weight and when they are split open they will be seen to be stained along the pith a dark bluish black, if the infection is heavy, or lighter shades of blue or black as the infection is lighter. The blackish colour is due to the large number of sclerotia which form along the fibers of the pith. These may be seen with the naked eye if the stem is split open (853).

The organism. Macrophomina phaseoli (Mabul.) Ashby. *Sclerotium bataticola* Taub.

The sclerotia are small, more or less round with numerous mycelial thread attachments. In general they are black, when seen alone, but the younger ones may be a light shade of brown, in which case the structure will be seen to be cellular in nature. See diagram. The mycelium appears to be of two sorts. One large and decidedly brown and the other small. The larger does not branch often whereas the smaller branches often and may bear numerous irregularities in shapes and sizes.



Diagrams illustrating *Macrophomina phaseoli* in sugar-cane stalks. Infection appears to come by the way of the roots and travel upwards. Many of the infected shoots are stunted with the nodes very short and the leaves yellowing before maturity.

A. Infected stalk showing the sclerotia in the pith.
B. Sclerotia magnified about 440 X.
C. Fragments of the mycelium.

Control. As has been previously stated (see under cotton and potato) the only control measure that appears to offer much hope of success is the use of organic manures before the planting is done.

Brown Leaf Spot of Cane

Host plants. Sugar cane varieties.

Geographic distribution. Appears confined to India and other Asiatic cane growing areas.

Appearance on the host plant. The disease receives its name from the colour of the necrotic areas which are brown with yellow bands around them. They are small being about one eight of an inch in size. As they age, the centers of the spots become straw coloured and the old spots appear as a series of rings.

The organism. Cercospora longipes Butl.

The conidiophores are longer than most of the species common on crop plants in India, being 100 to 200 by 4 microns. The conidia are tapering, 4 to 6 septate and measure from 40 to 80 by 5 microns.

Control. Resistant varieties appear to offer the most hope. At this time the disease does not appear to to be a limiting factor in cane growing.

Collar Rot of Sugar Cane

Host plants. Sacharum officinarum L.

Geographic distribution. Southern India. Petch (605) reported it in Ceylon.

Appearance on the host plant. The disease resembles red rot to a certain extent. The nodes near the roots are red. The pith for a few nodes will be watery while the upper part will be dry and pithy. Roots are also invaded.

The organism. Hendersonina sacchari Butler.

The fungus appears restricted to the red portion of the stem. It is inter and intracellular. All tissues are invaded. Chlamydospores are formed by portions of the mycelium becoming thickened and breaking off, later germinating and producing infection.

Pycnidia form on the old dead tissue and are found in stromatic masses as loculi. These masses rupture the epidermis and expose the tip which thus permits the escape of the spores. Most of the loculi are deeply sunken in the tissue of the stroma and irregular in shape and number.

The spores are of two kinds, similar to *Phomopsis*. Pycnospores are elongated with rounded ends, straight or occasionally curved, uniseptate or with one or two septa, brown in colour and measure 15 to 24 by 3.75 to 5 microns. The second type of spores are the scolecospores which are hyaline, filamentous, usually without septa but with many oil droplets. They are straight or slightly curved, tapering and measure 20 to 60 by 0.6 to 2 microns. Infection appears to take place by the roots or through wounds in the stem.

Control. General control measures have not been proposed but sanitation and rotation would be useful. See Butler (93)

Choudhury (113) reported that a bark disease of tea was caused by *Hendersonina theicola* Cke. in the Kangra Valley, Punjab.

Curvularia Leaf Spot of Sugar Cane

Hosts. *Sugar cane, sorghum, Citrus, wheat, maize, rice* and a number of other host plants.

Geographic distribution. It has been reported on plants in India, Japan, Africa, North America and Europe.

Appearance on the host plant. The spots are mostly along the midrib and at first look are very similar to those caused by *Colletotrichum falcatum*. They are surrounded by a red border which varies from 3-10 millimeters. The central portion is light gray varying in intensity of colour as the spots age, the central portion being darker than the margins. Here and there along the center of the spots will be seen gathering of mycelium that tend to make that portion darker and even to resemble acervuli of *Colletotrichum*. Examination of these spots with a hand lens will show that the acervuli-like spots are clusters of conidiophores.

These bear conidia at the top; singly, in pairs or sometimes in clusters.

Some times spots will be found, not along the midrib but on the lamina, and in that case they will be more or less rounded in shape. They are likely to have a narrower red border than those lying along the midrib. On rice it causes the black-kernel disease.

The organism. Curvularia lunata (Walker) Boedijn.

The organism has been known as *Acrothecium lunatum* Walker in literature but was renamed by Boedijn (72) as *Curvularia lunata.*

The conidiophores are erect and appear singly or in clusters of two or more. They are 100 to 200 microns long by 3 to 5 wide. The conidia are borne at the top, singly or more or less in whorls. See diagram. The conidia are from 1 to 4 septate with the typical number 3. They are usually curved, brown, and with the middle cell or cells larger. The end cells are often lighter in colour and may be nearly colourless. They measure from 18-19×8-12 microns.

Control. The leaf spot has been seen in the sugar cane fields at Allahabad for some seasons but it has appeared more severe this season (1947) than normal. It has also been observed on jowar and maize (901). The fungus was isolated from seed of jowar at the Agricultural Institute, Allahabad by Dr. W. N. Rice of Pyinmana, Burma, who proved its pathogenicity on seedlings.

Seed treatment is one way of helping control the disease. Copper carbonate, the mercury compounds, or some of the newer compounds such as Arasan will help keep down infection on the young plants. Ultimately it will require resistant varieties as the real control as later infections will not be controlled by seed treatment. Destruction of the old material from the previous seasons crop will also destroy the fungus in the leaves that it may not carry over.

Cytospora Disease of Sugar Cane

Hosts. So far the only host reported is *Saccharum officinarum.*

Geographic distribution. It has been reported in the United States, Japan and India. Abbott stated that it was in Louisiana in 1930 and by 1936 it was causing damage to the susceptible canes. Luthra, (Int. Bull. Pl. Prot X p. 262 (19367) reported in India in 1936.

Appearance on the host plant. The affected plants show a drying of the tops downward. The most severe form of the disease is a wilt. In the United States it is called a sheath rot and in India it has been named stem canker. These names evidently describe the appearance of the disease as seen under the differing conditions. Pycnidia form on the old dead litter and may develop on all of the above ground parts. The fungus becomes more virulent as the canes reach maturity. Luthra et al. (396) state that canes being preserved for seed, if buried in the ground, may be attacked and destroyed as the fungus can live in the soil as a parasite or a saprophyte.

The organism. Cytospora sacchari Butl.

The members of the genus are characterized by possessing a stroma which is covered but will be exposed by the covering layer rupturing and exposing a disc, which may be whitish and in which are one or more spores. Pycnospores are small, curved with rounded ends. Luthra et. al. (396) made isolations from a number of varieties, including Co. 312, Co. 313, Co. 323, Co. 371 and Co. 395, as well as varieties from separate sections of the Punjab and found them all identical. Artificial inoculations were 100 per cent successful with the infection spreading both above and below the point of inoculation. Symptoms would appear in 10-15 days and pycnidia in 17 days. Luthra, Sattar and Sandhu (398) found the temperature range to be from 5 to 40 C. with the optimum at 30 C. A study of the pH requirements showed that the growth stopped at 1.8 and 7.8. It was their belief that as the Punjab soils are all alkaline the fungus will not be likely to become serious.

Other Diseases of Sugar Cane

Subramaniam (758) reported *Helminthosporium halodes* on sugar cane in the Coimbatore Cane Breeding Station in 1934. McRae (362) had previously concluded that *H. sacchari* and *H. ocellum* were identical. This would indicate that at least two species of *Helminthosporium* are found on sugar cane. Butler (93) gives a record of *H. sacchari* on cane. He gives the measurements of the conidiophores as 100-190 × 5.5-7.5 microns. The spores are borne singly and fall readily. They are cylindrical, elliptical, thick walled, olive green in colour, 3-10 septate and 35-60 × 8.5-12 microns. Infected leaves show red spots which enlarge rapidly, and may, if they run together, form long streaks.

Cercospora kapkii was reported (96) as serious in Assam, especially in the region of Jorhat in 1938-39.

In 1939 Ramakrishnan (639) found a *Pythium* on sugar cane which he made a study of and believed it to be *Pythium debaryanum* Hesse. He was able to control it with bi-weekly irrigations of the soil with a $CuSO_4$ solution (1: 10,000). Later the same author (640) reported a *Fusarium moniliforme* (*Gibberella fujikuroi*) from sugar cane and bajra suffering from top rot.

Thirumalachar (781) reported an Ergot on sugar cane which is the first record of that fungus being observed on sugar cane in India.

The Common Diseases of Rice

A number of diseases of rice cause losses when the optimum conditions prevail for the pathogen. Among the more common diseases are the *Helminthosporium* leaf spot, bunt, sclerotial disease, false smut, black kernel disease, *Cercospora* leaf spot, rice blast.

Helminthosporium Disease of Rice

Host plant. Oryza sativa.

Geographic distribution. Probably general over India.

Appearance on the host plant. On the leaves the areas are oblong to oval, brownish with gray centers. On the culms and sheath they are longer, more irregular in shape and with larger centers.

The organism. Helminthosporium oryzae Breda de Baan. The conidiophores are found on the old diseased spots and possess the characteristic knee-bands so typical of the other species of *Helminthosporium.* They emerge from the stomata as from between the cells. They measure 70-175×5.6-7 microns. Conidia are few, somewhat scythe shaped, deep olive brown in colour and with from 1 to 6 septa.

Su (752) referred to *H. oryzae* as belonging to *Ophiobolus* and named the one on rice, *O. myabeanus* when he found the perfect stage.

Control. Sundararaman (759) found that 2% formalin solution, as a dip for the seeds for 15 minutes before planting, does no injury to germination and produces disease free seed. Mercury compounds and the more recent benzene and sulphur products are also expected to be valuable in control.

Bunt of Rice

Host plants. Species of *Oryza,* especially *Oryza sativa.*

Geographic distribution. It is known throughout the rice growing regions of south-east Asia and the U. S.

Appearance on the host plant. The disease is difficult to detect as the glumes often completely inclose the sori and there is no evidence of the smut. The sori are dark and are thus easily differentiated from the normal grain.

Organism. Tilletia horrida Tak.

The chlamydospores of the smut are round to somewhat elliptical, more or less opaque, light when young but black when mature, measure from 20 to 24 microns in diameter and bear on the walls somewhat curved scales which appear as a band around the outside. They do not germinate readily but when this occurs a septate promycelium is produced which bears a cluster of 10 to 20 needle shaped sporidia on the tip. These are 38 to 53 microns long and possess from 3 to 4 septa. The spore walls are sticky and adhere to any surface to which they may be blown and it is this character which makes the seed borne spores a menace.

Control. Copper carbonate appears to be best for the bunt of rice.

Sclerotial Disease of Rice

Host plants. Rice appears to be the only host plant.

Geographic distribution. Widely distributed.

Appearance on the host plant. The symptoms are various and no one appears to be constant. The most common symptom seems to be that of tillering together with a basal discoloration of the stem. The discoloration, however, may occur without the excessive tillering. Luthra and Sattar (395) found that a stem rot, in which the basal leaves and sheaths wither and turn brown and rot, is the most common symptom in the Punjab. Less common was the discoloration of the base of the stem without rotting, or the drying of the basal leaves. Presence of sclerotia on the stubble was always considered a certain sign that the disease had been present although there was no readily visible symptom.

The organism. Sclerotium oryzae Catt.

The fungus mycelium is more or less smoky in colour and is found both within and out side of the host cells. Sclerotia may be present within and without the host tissue and according to Shaw (684) resemble those of *Rhizoctonia solani* except in the larger size and more shiny appearance. They are black in colour, are from 1 to 10 mm. in diameter and arise from a white mycelium base that is also similar to that of *Rhizoctonia.*

Control. Control measures have not been worked out fully and no recommendations have been made. However, it should be possible to reduce the disease by the use of organic manures as is done with many other soil borne diseases.

False Smut of Rice

Host plants. The fungus has been found on other hosts than rice, as for example it also causes a false smut of maize.

Geographic distribution. It is widely distributed over the world, both the old and the new. It is found in many parts of India. Bengal, Bihar and Orissa of the northern part and Madras, Western Malabar and Tinnevelly in the south.

Appearance on the host plant. The first appearance of the fungus on the host plant is the enlarging of the ovaries into a nearly round, velvety green mass about twice the size of the ordinary grain. The young ovaries are attacked in the early stages of growth and the sclerotium is formed in 10 to 15 days. Saha (665) refers to the fact that the sclerotial bodies, which grow out of the ovary of the individual grain, appear above and between the glumes. The glumes are not changed, remaining closely attached to the center and lower part of the enlarged ovary.

The organism. Ustilaginoidea virens (cke.) Tak. Seth (681) states the mature spores are olive green in colour. When fully formed they are spherical and measure 4 to 6 microns. The optimum temperature for growth appears to be 26° C. The spores will remain viable for a period of 8 months.

Control. At the present time the best control appears to be rotation and the destruction of the bhusa that may contain the sclerotial bodies.

Black Kernel Disease of Rice

Host plants. It is not known whether other plants are affected by the same disease or not but it has been reported on rice.

Appearance on the host plant. The kernels are hard and a bright black in colour. Such kernels do not become broken in the polishing and thus differ from most of the coloured kernels found among the rice at harvest time.

The organism. Curvularia lunata Boedijn. The fungus is similar to *Helminthosporium sigmoideum* var *irregulare* which has been found capable of infecting the rice by Cralley and Tullis (139). *Curvularia lunata* was formerly *Acrothecium lanatum* but was transferred to *Curvularia* by Boedijn (72) when he found that it was not a good *Acrothecium*. At the same time *Helminthosporium inequalis Shear*, became *Curvularia inequalis.* A culture similar to *C. inequalis* was secured from rice and from juar seed at Allahabad. Saha (665) mentions that *Helminthosporium oryzae* has been found associated with the black kernel disease of rice.

The ovaries are susceptible to the attack of the fungus when in blossom. Rice straw is the apparent source of inoculum and on which the fungus over winters. Winds and water disseminate the spores during the blossoming period the next season.

The fungus receives its name from the fact that the spores are curved. They are typically three celled with the larger one in the middle. In mass the spores are a gray-black. The mycelium is a light brown when seen as single hypha.

Economically the disease is of greater importance than the usual small percentage of infected kernels would warrant as the dark kernels reduce the price of grain in the market. Infection is rarely more than one per cent.

Control. Avoid damp storage. Rotate the fields and destroy the stubble.

Cercospora Leaf Spot Disease of Rice

Host. Reported on rice in Burma but has not become wide-spread in India.

Geographic distribution. Has been reported in China, Japan, North America, Dutch East Indies, Brazil, Argentine, Puerto Rico and other parts of the world.

Appearance on the host plant. The diseased spots are chiefly on the leaves and are linear, elongate in shape. The centers of the spots are dark brown, fading toward the outer border. The shape of the lesion appears to depend on the susceptibility of the variety, being more linear as resistance increases.

The organism. Cercospora oryzae Myake.

There are at least six different races of the fungus. Chilton and Tullis (108) having recently added the sixth as a result of some studies carried on in Louisiana, U. S. A.

Control. There is much difference in the varietal susceptibility and thus the best control is likely to be that of selection for resistance.

Rice Blast

Host plants. Oryza sativa, Eleusine caracana, Panicum repens, P. ramosum, Setaria italica, Paspalum sanguinale, and *Triticum vulgare.*

Geographic distribution. In India it appears to be confined to the rice growing regions. It is also found in other parts of the world.

Appearance on the host plant. The disease appears on leaf blade and sheath as elongated grayish spots which vary in size from a millimeter or so to more than an inch in length and from one to several millimeters in width. The spots may coalesce and cover a large portion of the leaf and sheath. When the disease becomes epidemic the fields may show areas of plants which are grayish brown in appearance due to the large number of lesions.

The organism. Piricularia oryzae Cav.

The conidiophores and conidia of P. oryzae are smokey or ashy gray in colour. The conidia are borne singly or in scorpioid cymes at the tips of the branches. They are usually 3-celled and obclavate or pyriform in shape.

It appears that the conidia are the chief means of spread of the disease and they are produced mostly under high humidity. It has been reported that at least 93% of saturation was necessary for spore formation. At 88% no spores were formed.

At the Allahabad Agricultural Institute farm an epidemic occurred in the nursery plots in 1941. T. 136 was the first to show the disease and the first spots were recorded on June 24 by Mr. S. C. Bhatnagar who was in charge of the plots. When a reading was taken on July 12, T. 136 was heavily infected. Local varieties Malchi, Gulabkati, Jhalore and Bansmati were infected in that order, with Jhalore and Bansmati presenting only a few scattered spots.

Control. Control by direct measures is not easy Narasimhan (539) reported the control of P. oryzae on rice by dusting with sulphur flowers.

Aka (Ann. Phytopath. Soc. Japan IX pp. 223-235. 1939) found that rice from wet beds was more resistant to Piricularia than that from dry beds. When an analysis of the ash of the plants from wet and dry beds was made it was found that there was much more silicon in the cells from the wet fields. The added silicon in the cells made it much more difficult for the fungus to penetrate the host cells.

Thomas (797) reported that G. E. B. 24 was badly infected by *Piricularia,* in the uplands of Madras but that when crossed with Korangu samba, hybrids were secured which were more resistant that G. E. B. 24 and yielded from 10 to 18 per cent more than the original stock. From this it would appear that hybridization offers one of the best means of control.

Recently Anderson et. al. (26) reported that when *Helminthosporium oryzae* and *Piricularia oryzae* were grown together that the infection by *Piricularia* was reduced. However they do not suggest that this is a means of reduction of infection but rather a substitution.

*†

The Sclerotium Disease of Rice

Host plants. *Oryza sativa* L.

Geographic distribution. Widely distributed.

Appearance on the host plant. One of the first evidences of the disease in the field is the excessive tillering which takes place at the base of the infected culms. These turn yellow and die and may or may not bear a grain. Any grain produced is light and poorly developed. In some cases there is no evidence of the disease until the crop is well advanced. Tillering is not observed in all countries.

The organism. *Sclerotium oryzae* Catt.

The first symptoms of the presence of the fungus itself are the small black lesions on the outer sheath. If this portion is split longitudinally, the basal portion will be found full of dark grayish hyphae and a transverse section will show hyphae and sclerotia. The sclerotia are black, 1/10 mm. in diameter, glistening and arising from a white mycelium very similar to that of *Rhizoctonia.*

Control. Probably the best control measures will be resistant varieties.

THE MORE IMPORTANT COMMON DISEASES OF COTTON

Cotton is grown over much of India and many varieties of the crop have been brought in from outside. With these have come some very serious diseases. Among the diseases that are serious on cotton at this time may be listed, wilt caused by *Fusarium vasinfectum*, *Sclerotium bataticola*, and *Sclerotium rolfsii*; anthracnose, cotton root rot and *Cercospora* leaf spot.

Cotton Wilt

Hosts. Kulkarni (355) found at least three organisms that were associated with the wilt of cotton. Each possessed a wide range of host plants and as a result the cause of cotton wilt can be said to be due to fungi that are common on many plants. *Rhizoctonia bataticola*, *Sclerotium rolfsii* and *Fusarium vasinfectum*. *Fusarium vasinfectum* is the most commonly met with and considered the most important of the three from the stand point of cotton wilt.

Geographic distribution. World-wide.

Appearance on the host plant. Wilt usually occurs on scattered plants and not on closely associated plants. This makes it more difficult to detect the diseased plants than in the case of some of the diseases. like root rot, where an area may all be infected alike.

Wilt will first be observed as a tendency to wilt during the day but recuperate over the night. This will continue until the plant fails to recover and eventually dies. Wilted plants may be identified by cutting into the wood or peeling back the bark. If the plants are infected by the wilt organism the wood beneath the bark will be seen to be brown, may be wet and the discoloured portion will be in the form of a ring. This discoloured portion is the vascular ring and a section of the infected tissues will show the fungus hyphae in the vascular tubes (Xylem vessels).

The organism. Fusarium vasinfectum Atk.

The organism was first described by Atkinson in 1892. E. F. Smith in 1899, confused the *Fusarium* with the *Ascomycete, Neocosmospora vasinfecta.* In 1910 Butler corrected the idea of the parasitism of the *Ascomycete* and in 1912 proved *F. vasinfectum* as the cause of cotton wilt.

Fusarium vasinfectum is one of the imperfect fungi with no well known perfect stage. The macroconidia are somewhat narrow, mostly 3-septate, 27-28 ×3-3.75 microns. Thick walled chlamydospores are found at the tips of, or in the hyphae strands, and these are returned to the soil as the cotton stalks decay.

The conidia are air borne and they, together with the chlamydospores, are responsible for the spread of the disease or of the carrying over of the fungus from one season to another. Any agent that can move the soil which bears the chlamydospores will act as a distributing force.

The fungus grows best at about 28-30°C. It appears to do better on light soils than on heavy.

For the description of *Rhizoctonia bataticola* see under potato.

For the description of *Sclerotium rolfsii* see under ground nut.

Control. As the cotton wilt organisms may live for years in the soil, the control does not include a rotation in the ordinary sense. It is not seed borne. It is found more severe on plants that are deficient in potash. Potash deficiency is manifested by rusty spots appearing on the leaves. These are not real rust but only in appearance. Enough potassium—containing fertilizer to correct the deficiency will do much to eliminate the disease.

Subramanian (757) states that the cotton wilt will not remain alive in unsterilized soil for long. He considers that microbiological antagonism of the saprophytic forms in the soil is responsible for the destruction of the pathogen.

Anthracnose of Cotton

Hosts. Species of *Gossypium.*

Geographic distribution. General where cotton is grown.

Appearance on the host plant. On the bolls the spots are more or less circular with depressed centers. Because of the characteristic pink masses of spores that form on the infected areas the disease is often referred to as "pink boll rot". Dastur (157) described the disease on cotton in the Central Provinces but at that time apparently considered it a new species. He described the symptoms on bracts, bolls, lint and seed.

On the bracts the spots appear circular in shape, brown or black in colour, water soaked and are visible on both sides of the bracts. The fungus may pass on to the boll and if it reaches the boll base the boll usually falls. Dastur states that he did not see acervuli on the bracts.

On the bolls the infection may take place at all stages. The first symptoms are small water soaked spots, dark in colour with slightly depressed centers. The centers darken with age, dry, shrivel and die. Small infected bolls fall off. A badly diseased boll may become mummied. If, however, it is diseased on one side only, they are likely to grow one sided. If the tip of the boll is infected it will split. If infection takes place through the pistillary end the whole boll may become diseased from top to base. The bolls split open and the contents turn black and minute black dots appear. These are acervuli of the fungus. If conditions are optimum for spore formation these spots may become pink. This pink colour is due to the spore masses. Lint in the infected portion does not emerge from the bolls but remains stuck together although the healthy portion may open and the lint emerge more or less normally.

Seedling infection may start from root or cotyledon. When root infection occurs a damping off may take place. The first lesions may be water soaked, elongated, reddish or brownish and may be on radicle or collar. If the lesions spread so that the radicle is encircled the plant is likely to collapse and fall over. Infection on older plants are likely to become cankerous with the cortical tissue sloughing off and leaving the tissue beneath exposed.

The presence of acervuli is the best macroscopic means of identification, especially in the case of cotyledon infection.

The organism. Dastur (157) named the organism *Colletotrichum indicum.* The acervuli are bristly and black when occurring on the stems, but are more inclined to be pink coloured, globular, oily in appearance and formed in compact rings when infection occurs on the bolls.

Setae are thick walled and develop from basal stromatic cells. They are multiseptate, varying from 1-7, lighter at the base and more or less sharp pointed. They measure 76.5-255 × 3.8-7.6 microns. The conidiophores are hyaline to sub hyaline, finger shaped, slightly curved and broadly rounded at the apex. They measure 7.7-13 × 1.6-2.7 microns.

The conidia are falcate in shape, falling soon after being formed. They measure 20-22.5 × 2.5 microns.

Control. The disease is largely seed borne and tests with seed delinting with sulphuric acid gave over 50 per cent reduction in the death rate over the untreated.

Cotton Root Rot

Hosts Extremely wide range.

Geographic distribution. World-wide.

Appearance on the host plant. Cotton root rot has some of the same characters that are found in wilt. But in the former case there is decay of the roots and in pure wilt there may be no evident decay until after the plants are dead. Prasad (623) considered that *Rhizoctonia bataticola* (*Macrophomina phaseoli*) and a species or *Fusarium* (not *Fusarium vasinfectum*) were most commonly associated with the disease with *R. bataticola* being the most active. It might be well to mention that the fungi causing the cotton root rot in India are not the same as that causing the root rotting in the United States. The so-called Texas Root Rot in the United States is caused by *Phymatotrichum omnivorum* (Shear) Duggar.

Plants affected with root rot show symptoms of yellowing of the leaves, dropping of the bolls before mature, are easily removed from the soil and usually show blackening at the ground line. If pulled up they come out easily and the major roots are decayed, blackened and often bear many sclerotia of the fungus over the decayed portion.

The diseased plants are likely to occur in definite areas which differs from wilt as the plants affected with the wilt organism are much more likely to be scattered.

Plyman (613) stated that seedling blight may be produced by the fungus under some circumstances. This was especially true of the late planted cotton.

The organism. Sclerotium bataticola Taub. has been suggested by Henson and Valleau (281) as the correct name. They state that as the fungus appears to be much more like the *Ascomycetes* than the *Basidiomycetes* it should be called *Sclerotium* rather than *Rhizoctonia. Basidiospores* have not been found and pycnidia are not known in the *Basidiomycetes.* No clamp connections have been found. These are also characters if the *Basidiomycetes.* (Clamp connections are the conjugation tubes that connect the cells of certain *Besidiomycetes* when fusion takes place).

The perfect stage of *Sclerotium bataticola* is supposed to be one of the *Ascomycetes.* The *Macrophomina* being the pycnidial stage. Ashby (32) concluded, after careful studies, that *Macrophomina phaseoli* Maubl., *M. corchori* Saw, *M. cajani* Syd. and Butl., *Sclerotium bataticola* Taub., *Rhizoctonia lamel-*

lifera Small, *R. bataticola* (*Taub.*) Butl. *Dothiorella cajani* Syd. and Butl. and *Macrophomina philippinensis* Petr. are all one and should be called *Macrophomina phaseoli* (Maubl.) Ashby. Thus referring back to the first of this discussion the correct names should be;

Sclerotium bataticola Taub. for the sterile stage and
Macrophomina phaseoli (*Maubl.*) Ashby for the pycnidial stage.

Haigh (258) divided the group into three classes, according to the size of the sclerotial bodies, as follows;

C. group. sclerotia measurements up to 120 microns.
B. group. sclerotia measurements up to 200 microns.
A. group. sclerotia above 200 microns.

Vasudeva and Ashrof (872) found that the optimum temperature for *Rhizoctonia solani* was 35°C. where as that for *Macrophomina phaseoli* was between 20 and 40 a much wider active range. The pH. range of each was wide. They would tolerate acidity and alkalinity from 2.4 to 9.2. Heating at 65°C. for 5 sec. killed *R. solani*. *M. phaseoli* required 68°C. for 5 sec. to kill.

Prasad (624) found that when a species of *Fusarium* was associated with *Rhizoctonia* that greater virulence was displayed than when either was considered alone. See *Pythium* blight of wheat. Fusarium alone would produce 19 per cent infection. Rhizoctonia alone would produce 26 per cent infection. But when mixed in equal percentages they produced 80 per cent infection among the plants.

Control. Sundaraman found that the pycnospores would retain their vitality for as much as 14 months. When he used loam at the rate of 1000 pounds to an acre the infection was reduced about half.

Likhite and Kulkarni (380) report that well water, which had a pH of 8 produced more root rot by *R. bataticola* than was found in fields which received only rain. A humidity of 30 per cent at 40° C. favoured the parasite. In the Punjab (380) M. *phaseoli* was more severe on early planted cotton than on later planted. Cotton planted May 7 yielded 123 lbs. to the acre but that sown June 25, yielded 369-430 pounds to the acre. Luthra and Vasudeva (399) report that when jowar was interplanted with cotton the dead cotton plants numbered only 3-4 per cent as compared to 68 per cent of dead plants in the pure cotton. Vasudeva (873) in a later report stated that when they used American cottons LSS and KT 25 interplanted with *Phaseolus aconitifolius* the diseased plants were reduced from 63 to 1 and 46 to 3 in two trials. When Indian cottons were interplanted with the same legume the disease incidence was reduced from 55 to 2 and 52 to 2 per cent.

From the above it would appear that there is much to be done in the field of control of cotton root rot. Some things appear possible at this time.

1. Plant after the middle of June.
2. Interplanting appears to reduce the pathogenicity of the organism. However we do not know about the yield in this case.
3. Liming the soil appears to help.

Cercospora Spot of Cotton

Hosts. Cotton.

Geographic distribution. It has been reported from a number of the cotton growing areas of the world. Probably general.

Appearance on the host plant. On the leaves the spots are nearly round, gray centered with a darker

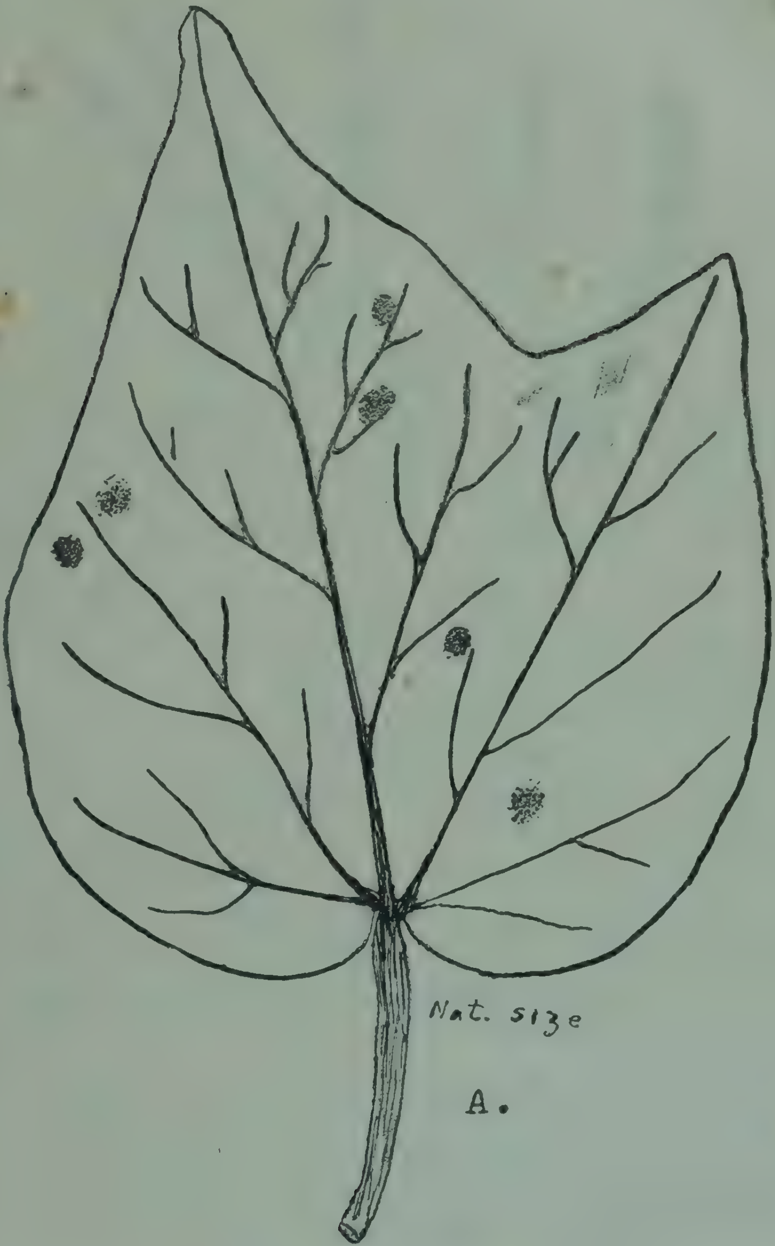
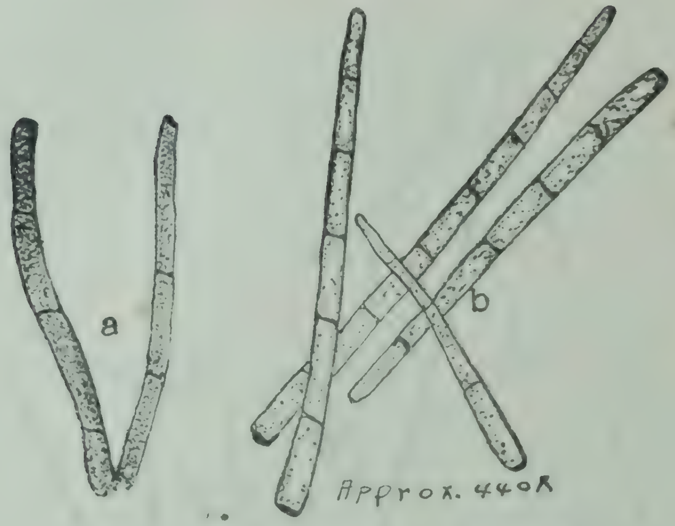


Diagram of *Cercospora gossypina* on cotton leaf.
A. Leaf showing the spots.



B. a. Conidiophores b. Conidia of *Cercospora gossypina.*

border. They may appear on both sides of the leaves and there are usually more conidia on the lower surface than on the upper.

The organism. Micosphaerella gossypina (Atk.) Earle. *Cercospora gossypina* Cke.

The perithecia are ovate, black, partly sunken in the tissue and measure 60-70 × 65-91 microns. The asci are sub cylindric and measure 8-10 × 40-45 microns. The ascospores are elliptic with slight constrictions at the septa. They measure 3-4 × 15-18 microns.

The conidia (see drawing) are attenuate (tapered) above and 5-7 septate. They are hyaline and 70-100 × 3 microns.

Control. The disease is not serious and most seasons is not of importance. Destruction of the old debris so that none of the mycelium or spores could be carried over will aid in the control.

CHAPTER X

THE MORE COMMON DISEASES OF THE LEGUMES IN NORTHERN INDIA

SOME OF THE MORE COMMON DISEASES OF SUNN HEMP

Among the diseases to be found on sunn hemp is wilt caused by one or more of at least three fungi. *Fusarium vasinfectum, Rhizoctonia solani* or *Macrophomina phaseoli* may cause wilt, either alone or in combination.

Crotalaria Wilt.

Host plants. Species of *Crotalaria* are only a few of the many hosts that are attacked by the organism causing the wilt of *Crotalaria juncea.*

Geographic distribution. The fungi causing the wilt are wide-spread over India and the world. They will probably be found in any soil which has been cropped to legumes, vegetable crops and forage crops.

Appearance on the host plant. One of the first symptoms of the wilting is observed in the leaves. They gradually wither, droop, turn brown and within a short time the whole plant dies. Sometimes when only one side of the plant is infected, branches on that side only will wilt. In mature plants the wilting begins at the tip followed by defoliation.

On the diseased portions of the branches sporodochia are seen forming a pinkish mass. This indicates the organism is a member of the genus *Fusarium.* This fungus is a vascular parasite and may be found in the xylem vessels. The vascular tissue is discoloured and the discoloration may be traced from the root to stem. Early infections are likely to be confined to the root tips.

The organisms. Fusarium vasinfectium Atk. (See under cotton) *Rhizoctonia solani* Kuhn (See under Potato) *Neocosmospora vasinfectum* Smith.

Fusarium vasinfectum is a vascular parasite and may be found in the xylem vessels. Discoloration of the vascular tissue is one of the symptoms of the *Fusarium* parasite. Under optimum moisture and temperature the *Fusarium* will fruit and the characteristic pink spore masses form over the diseased portions of the stem surface. These spores are spread by water, cultivating tools and diseased plant parts and are a source of infection to the other plants. The fungus may even invade the pods and seeds and at thrashing time the other seeds may become contaminated with the spores.

Rhizoctonia solani is a root rotting parasite and while it may invade the upper portion of the host it is less likely to than *Fusarium*. Symptoms may be very much the same. Examination of the roots will show, not masses of pink spores, as in the case of *Fusarium vasinfectum*, but small black sclerotial bodies on the diseased roots. This difference makes differentiation relatively easy.

Neocosmospora vasinfecta which belongs to the tribe *Nectrieae*, forms perithecia which are red in colour with typically two-celled, brown ascocpores. Mitra (492) reported that the fungus caused the death of a high percentage of sann hemp plants in tests conducted at Pusa in 1935.

Control. Control of root rotting caused by such cosmopolitan fungi as the ones mentioned is a real problem. Rotation to cereals will help to inhibit but not prevent, the fungi from finding host plants. Perhaps the best control which is being suggested at this time is a heavy application of barnyard manure, plowed under and permitted to rot in the soil. This will build up the soil bacterial flora which is antagonistic to the fungi and thus hold them in check.

Uppal and Kulkarni (854) found the optimum temperature for the fungus to be 25-30°C. A low moisture content was favourable but the temperature was most important. They recommend a resistant variety D-IX which has maintained its resistance for a number of years.

Gray Blight of Sann Hemp. (Anthracnose)

Hosts. Crotalaria juncea.

Geographic distribution. Reported on sann hemp in India.

Appearance on the host plant. The first appearance of the disease is on the cotyledons. The fungus then spreads to the stem and the growing point. Later the infected spots turn brown and may be found on all parts except the underground portions. The fungus spreads rapidly and within 48 hours the acervuli are formed in the infected areas with the production of large masses of spores. The growing point is soon killed and the young plant dies. In the case of older plants the spots appear on one side of the leaf first but gradually enlarge and extend to the opposite side of the leaf. These are grayish brown lesion rounded or angular. The spots coalesce and may cover a large area. The spots may also form on the midrib.

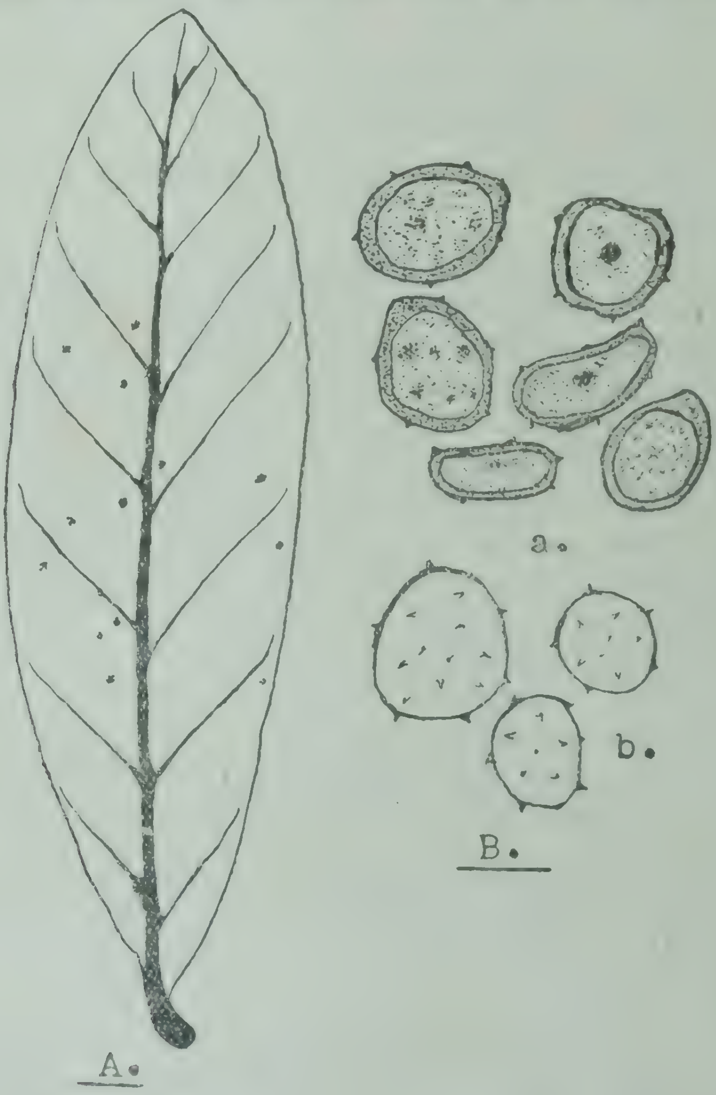
The organism. Colletorrichum curvatum Briant and Martyn.

The fungus was first reported on *Crotalaria juncea* by Briant and Martyn (81) who found it in Trinadad. Mitra (492) reported in on the same host plant in India.

Acervuli form in abundence on the diseased areas of the host plant. They are white or a faint pinkish in colour and consist of simple, erect, closely packed conidiophores and setae.

The setae are brown to dark brown, septate, tapering toward the tip, the base swollen. They form among the conidiophores and measure 66-140×4-6 microns, average dimentions being 104.64×4.98 microns.

The conidia are one celled, hyaline, falcate and acute at the end. In size they are 15-24×3-4 microns



Diagrams illustrating the rust on Sunn hemp.

A. Leaf with uredo sori B. Uredospores a. In cross section b. in outline.

with the average being 18.65×3.03 microns (Mitra 492). These vary slightly from those of Briant and Martyn.

Control. Control measures have not been worked out fully as yet. Clean seed is one means since the organism is seed borne.

Rust of Sunn Hemp (*Urmyces decoratus* Syd.)

Hosts. It appears confined to sunn hemp.

Geographic distribution. It has been reported in various parts of India but does not appear in Arthur's book of rusts (31) so it would appear from that it is not widely distributed.

Appearance on the host plant. It appears as small, irregularly shaped spots which are golden brown in colour. They may be on both surfaces of the leaf but more common on the upper surface. Uredosori are found on the leaves during the growing season. They are disseminated by wind and rain.

The organism. Uromyces decoratus Syd.

Uredospores are golden brown in colour, fairly thick walled and with few small spines. Teleutospores are dark brown, single celled and borne in sori similar to the uredospores.

Control. At present no special control measures have been worked out. It has not been found serious enough to give alarm as yet.

THE MORE COMMON DISEASES OF ALFALFA IN NORTHERN INDIA

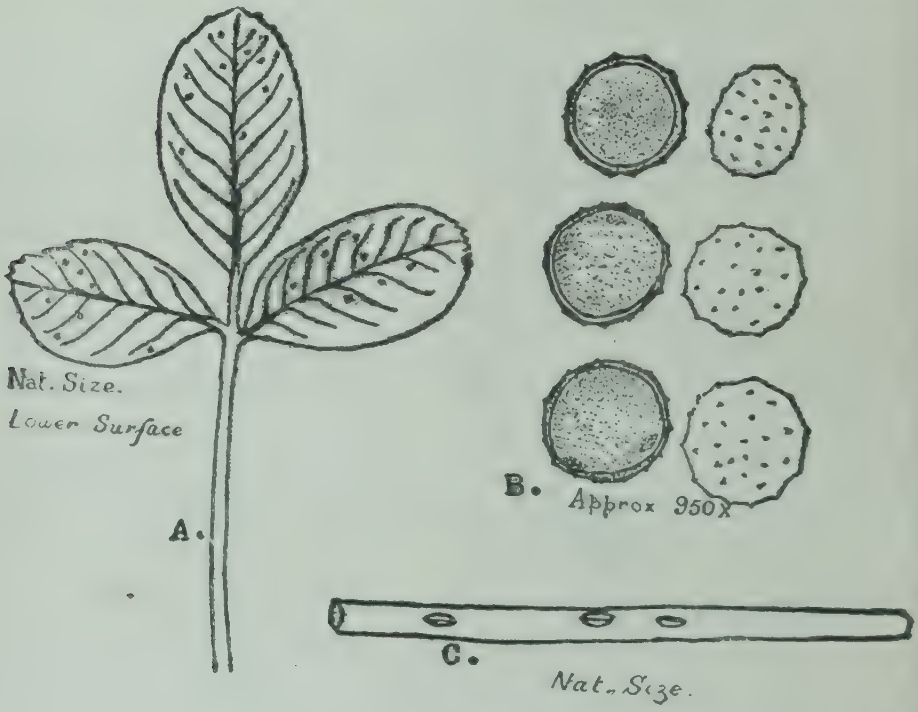
Alfalfa is not a very important crop over India as a whole but it is grown as a forage crop in some areas. There a few diseases that are of interest may be found on the crop. Probably the most important is alfalfa rust. Downy mildew was common in the Allahabad region in 1946-47 and did some damage. *Cercospora* leaf spot and one or two other fungi made their appearance as minor diseases.

Alfalfa Rust

Hosts. Has been reported on various species of *Medicago* and *Trifolium.*

Geographic distribution. It has been reported over the world in practically all regions where alfalfa is grown.

Appearance on the host plant. The uredosori are commonly brown and nearly circular in shape on the leaves. On the stems they are more likely to be elongated in the direction of the long axis of the stem.



Rust on alfalfa leaf and stem, and the spores.

A. Diagram of a leaf showing the lower surface with the sori scattered between the veins.

B. Uredospores in cross section and outline.

C. Diagram of a stem showing the sori. Note the elongated shape as compared with the round ones on the leaf.

The teleutosori are somewhat darker than the uredosori. Uredosori are on both sides of the leaves but more likely to be on the undersurface. Infections start with the lower leaves but as the season advances there is more and more infection on the upper leaves. Early infected leaves may fall and thus deprive the plant of that much foliage.

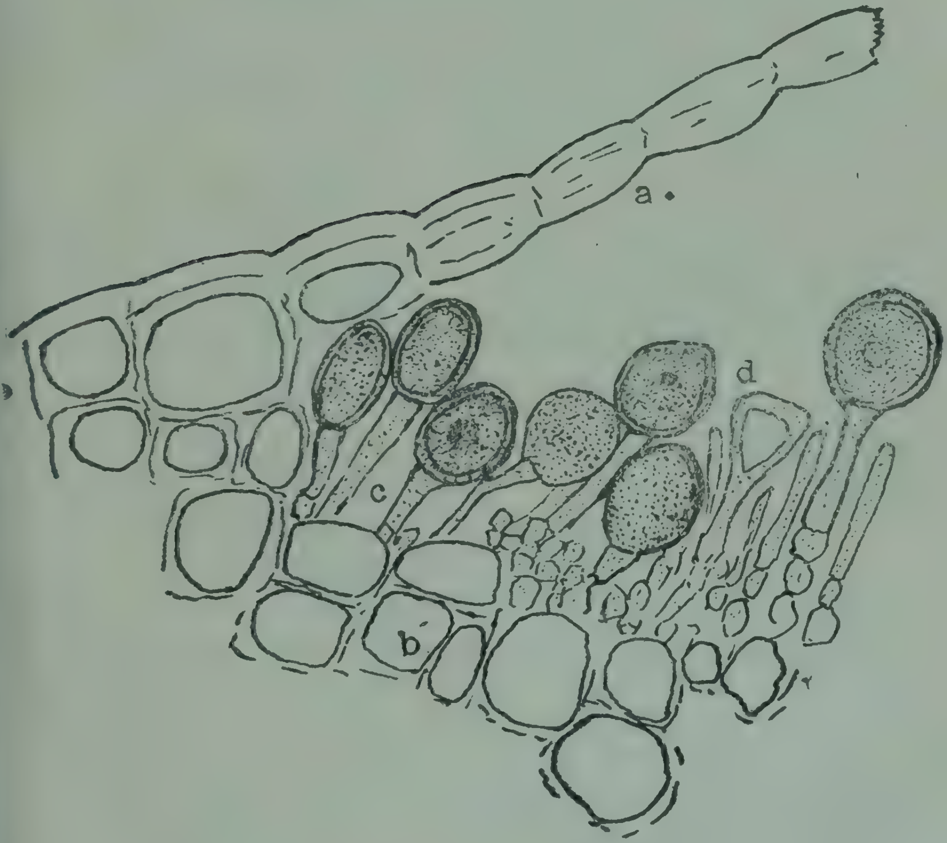


Diagram of a cross section through a teleutosorus of alfalfa rust.

a. Epidermis b. Mesophyll tissue.

c. Teleutophore d. Teloutospore.

The organism. Uromyces striatus Schroet. Arthur (31) suggests a new combination of *Uromyces striatus medicaginis* (Pass) Arthur. Uredospores are globoid, 16-2×18-23 microns. The walls are a golden brown, spiny (see Figure) with 3 to 4 germ pores. The Teleutospores are darker and fro globoid to illipsoid. (see Figure). They measure 15-20×19-24 microns. There is a hyaline papilla visible over the pore. The walls are verrucose rather than spiny. The rust is not serious in the region of Allahabad but appeared in epiphytotic form in 1947. Humidity was considerably above the average for the winter season of 1946-1947 and this was no doubt responsible for the heavy attack on the alfalfa.

Control. It is not of sufficient seriousness to warrant concern here and no disease control measures have been worked out. About the only control measure suggested is to harvest frequently so that the leaves may not fall to the ground and thus over winter.

Downy Mildew of Alfalfa

Hosts. Species of *Medicago, Glycene* and *Trifolium.*

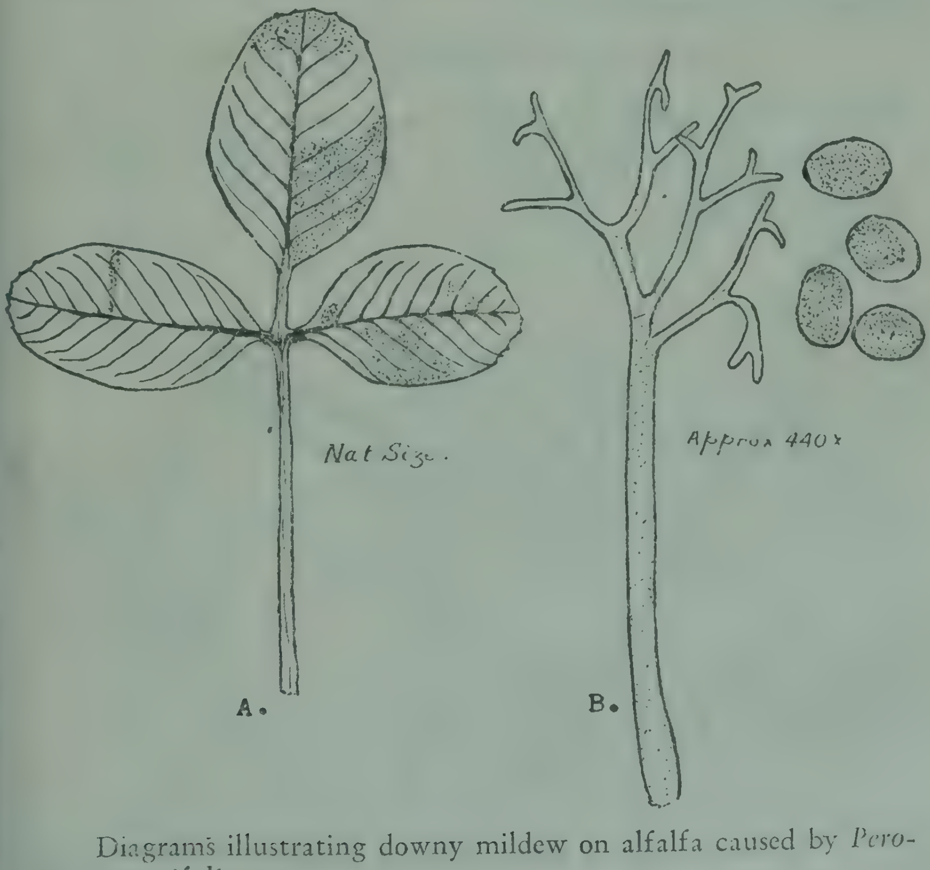
Geographic distribution. The world over.

Appearance on the host plant. On the leaves irregular light yellowish spots appear that are varied in shape and size. They vary from one to several mm and may include the whole leaflet. The outer margin of the area is frequently lighter in colour than the middle portion sometimes being almost white. On the under surface the central portion may become a faint purplish gray. These are the conidiophores and conidia.

The diseased leaves may be readily recognized by the light coloured areas on the leaflets which may later dry and the leaflet curl at the tip. Sometimes the spots take on a reddish tinge, although this is not a reliable symptom.

The organism. Peronospora trifoliorum de Bary. Although the host range includes other than alfalfa, such as soy bean, there is some confusion as to the organism that is found on the soy bean. Butler and Bisby (96) record *P. trifoliorum* on soy beans in Kashmir but in the United States and Manchuria it is considered to be a different species and called *P. manchurica* (Naoum) Gaumann.

The conidiophores (see Figure) are 360 to 600 by 9 to 11 microns, several times branched with the secondary branches likely to be curved or drooping. The conidia are globose to broadly elliptic and measure from 15-18×20-36 microns. The oospores are glob-



Diagrams illustrating downy mildew on alfalfa caused by *Peronospora trifoliorum.*
A. leaf with mildewed areas. B. conidiophore and conidia of the fungus.

ose, smooth, light brown in colour and from 24-30 microns in diameter.

Melhus and Patel (471) in a study of the fungus found that the optimum temperature for growth is 15° C-22°C with a high humidity essential. In 1946-47 the temperature and humidity at Allahabad were optimum and as a result the heaviest attack of downy mildew was recorded on alfalfa in recent years.

Control. From season to season the disease is not serious. Control measures have not been worked out but resistant varieties have been developed in some parts of the world and where climatic conditions are favourable for the fungus it is likely that they would be the solution to the problem.

Cercospora Leaf Spot of Alfalfa.

Hosts: Reported on alfalfa and some of the clovers.

Geographic distribution. Wide-spread where alfalfa is grown.

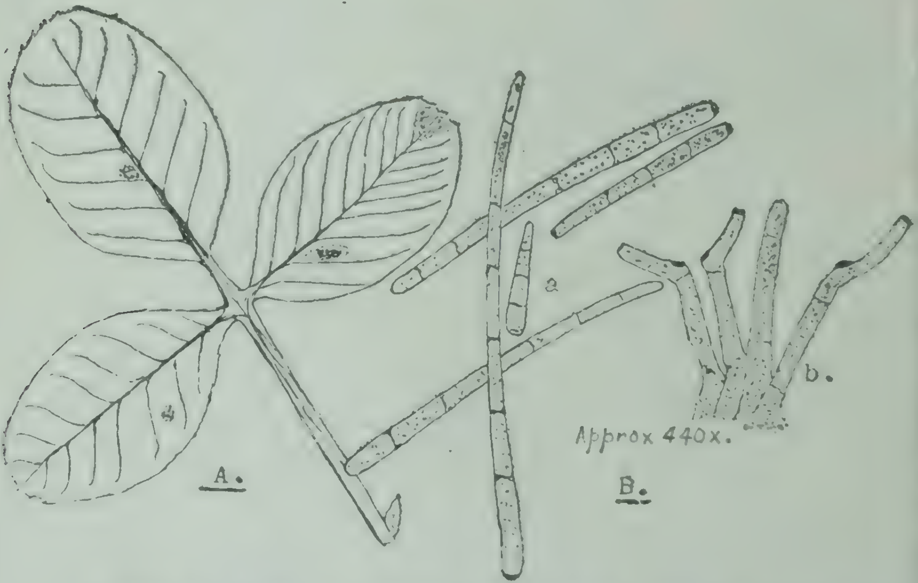


Diagram of a leaf of lucerne infected with *Cercospora medicaginis* E. & E.

A. Infected leaf B. a. conidia b. conidiophores.

Appearance on the host plant. The spots appear on the leaves as irregular, dark, smoky or almost black spots which vary from 0.5-5 mm in diameter. They may appear almost zonate but are indefinite around the margins.

The organism. Cercospora medicaginis E. & E.

The conidiophores are short with rather sharp knee bends where conidia have fromed and fallen. The conidial scars are distinct. The conidiophores are hyaline to subhyaline but become darker as they age. Because of the shortness and the light colour it is difficult to see the clumps without a fairly strong lens. Conidia are tapering, septate with 3-6 septa and measure from 40-60 microns. The conidiophores measure 35-45 × 4-5 microns.

Control. It is not serious so that it is probable that only under very optimum conditions the pathogen becomes serious. But early cutting and rapid harvesting of the infected leaves will aid in taking away the source of inoculum.

Alternaria Leaf Spot of Alfalfa

This fungus was observed causing a leaf spot of alfalfa this year for the first time in the vicinity of Allahabad. It was associated with a leaf spot caused by *Cercospora medicaginis* and *Peronospora trifoliorum* but it was not observed in the same spots. The spots were of two kinds. One that appeared distinctly zonate, like the frog eye spots of *A. solani* on potatoes, and the other undefined in which case it appeared not to be the pathogen. It was the only fungus that could be found in the spots with the distinct zones in the center and there appeared to be definitely pathogenic in the field. It is being studied further.

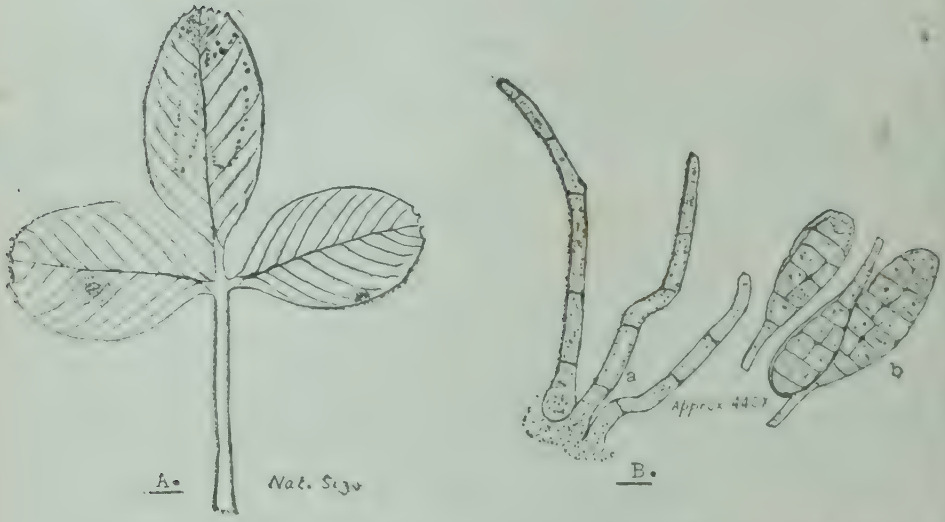


Diagram of a leaf of lucerne infected with a species of *Alternaria.*

A. Leaf showing typically zonate spots.
B. a. Conidiophores b. Conidia.

An Anthracnose of alfalfa

Hosts. Lucerne.

Geographic distribution. Anthracnose of alfalfa is widely known over Europe and the Americas. In Europe the disease has been caused by *Colltotrichum trifolii* but this organism has not been recorded in India.

Appearance on the host plant. On the agricultural Institute Farm in 1947 an anthracnose was observed on the lucerne plants that infected leaves, stems and inflorescence. The spots on the leaves and stems are dark gray to brown. Darker on the petioles and stems than on the leaves. Spots on the leaves measured up to 2 mm. in diameter where as on the stems they may be from 5 to 20 mm. in length and 3 to 10 mm. in diameter. The infected areas are usually lighter toward the center but might be dark because of the number of accrvuli present. Blossom blight was also evident in many cases, the inflorescence being distort-

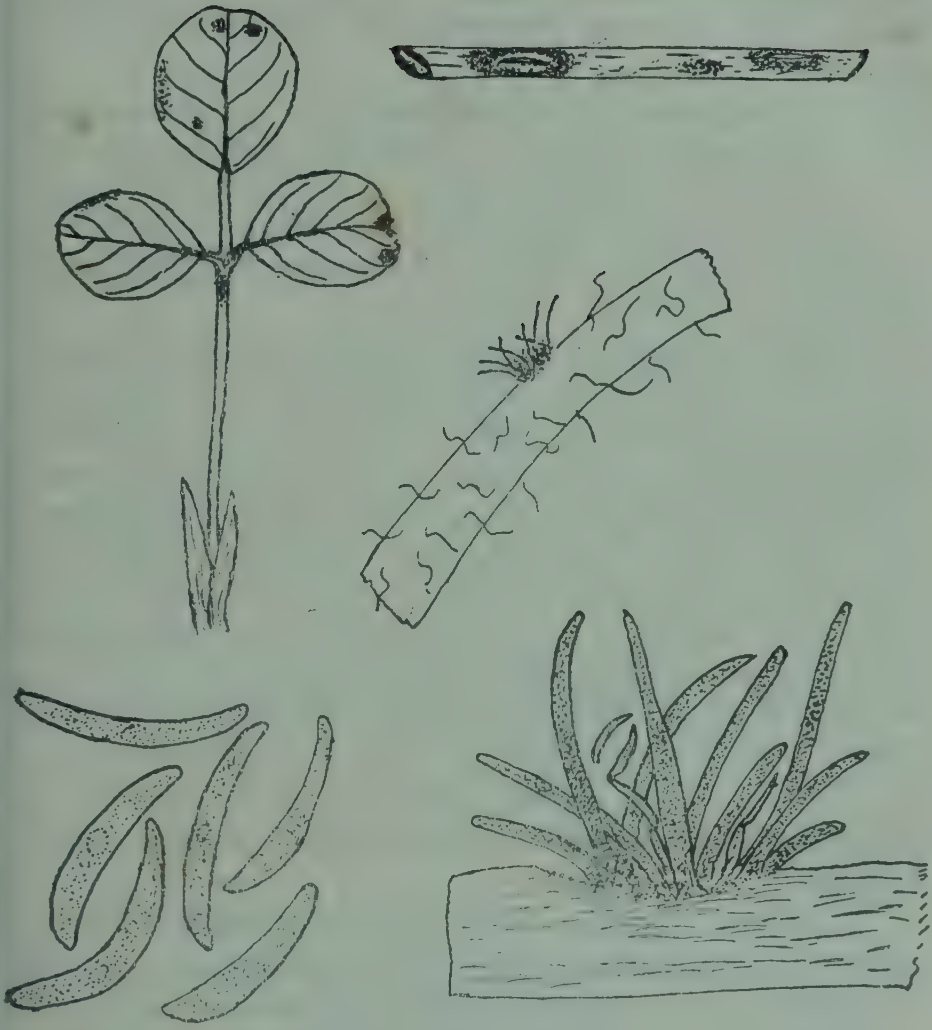


Diagram illustrating a species of *Colletotrichum* observed on alfalfa on the Institute farm during the 1947 season.

A. Typical leaf infection.
B. Typical stem infection.
C. Infection on a portion of the inflorescence.
D. Cross section of an acervulus with setae and conidia.
E. Conidia enlarged.

ed and twisted and invariably sterile.. Under a hand lens the acervuli were observed to be small but in large numbers.

The organism. Colletotrichum sp. The acervuli are small, nearly circular with prominent, nearly straight setae which measure 1½-5×32-80 microns, average 2.68×45. Conidia are slightly curved, rounded at the tips, hyaline and measure 4-5¼×23-34 (average 4.66×28.8) microns.

McDonald (429) reported *Colletotrichum trifolii* on lucerne in Kenya in 1926 and it had been reported in South Africa before that. (429) But the characteristics as given for *C. trifoii* do not correspond closely enough to those found on the lucerne at Allahabad to justify calling them the same. *Kabatiella caulivara* (Kuchn.) Karak was studied by Miss Sampson (666) on clover and alfalfa in England gut the description of the fungus causing blossom blight of clover shows it to have sickle shaped spores and that does not agree with the spore shape of the one found at Allahabad.

The organism is still under study at Allahabad. This constitutes merely a report on observations made to date.

Control: None is being suggested which is backed by experimental evidence. But experience gained with other similar legume anthracnose diseases would lead to the suggestion that resistant varieties will be the ultimate answer if it proves serious over a period of years.

The More Common Diseases of Arhar in Northern India.

The most serious disease found on arhar is that caused by *Fusarium*. It is the same fungus that causes the wilt of sunn hemp and is also associated with the wilt of other crops, such as cotton. A *Diplodia* and a *Cercospora*.

Fusarium Wilt of Arhar (Cajanus indicus Spreng.)

Hosts: A wide range of host plants but principally among the legumes. The fact that there have been synonyms which are now recognized makes the host range larger than at first thought.

Geographic distribution. Over India and probably in other countries as well.

Appearance on the host plant. The first symptoms will be a withering of the tops and probably a yellowing of the leaves even though there may be humidity in the air and moisture in the soil. The plants may be pulled from the soil some what more readily than normal ones and examination of the roots will show them blackened and often with streaks running up the stem. When the bark is removed and the wood sectioned dark stained portions are observed in the vascular portion of the tissue. The disease may appear at any time but is more pronounced and prominent during the colder months of the year. The small plants may die and not be noticed but if a large one begins to wilt it is seen from a considerable distance. It is often possible to see the mycelium of the fungus on the diseased roots if the soil is moist.

The organism. Fusarium udum Butl. Fusarium vasinfectum Atk.

In 1926 (94) Butler concluded that *F. udum*, *F. cubense* and *F. vasinfectum* might well be called the same organism. But in 1940 Padwick (572) determined that *F. udum* and *F. vasinfectum* are not the same but are distinct species. Wollenweber (938) concluded that the pigeon pea in India is attacked by two species of *Fusarium* with *F. udum* being the more serious. *Fusarium vasinfectum* has been found on arhar in a number of places in India and must be considered as one of the wilt producing organisms.

The fungus penetrates the vascular tissue of the plants and a section of the woody portion will show many hyphal threads in the large xylem vessels as well as other tissues. See illustration under tomato. It is the plugging of the vessels that causes the plant to wilt.

Infection appears to take place through the lateral roots as they are among the first tissues to be blackened. Infection, according to Butler (93) takes place through the fine roots and not usually on the larger roots by injuries. He states that there is no evidence that it ever takes place above ground. Observations made at Allahabad substantiate this evidence.

The microconidia are borne at the tip of the fungus hyphae and thrown off as fast as they are formed. They measure 5-15×2-4 microns. Under moist conditions they may remain adhering to the tip of the conidiophore until several may collect. These are sometimes referred to as the *Cephalosporium* stage of the fungus. The genus being set up on the character of spores being held together in a slimy mass.

The macroconidia are 15-50 × 3-5 microns with from 3 to 5 septa. These are free and do not adhere in a slimy mass. It is the Fusarium stage of the fungus. These are easy to differentiate from the microspores because of the size and also because they are pointed at each end and curved like a new moon.

Control. Soil borne diseases are not easy to control. As has been mentioned in the section on root diseases, organic manures appear among the best of the control measures. McRae in 1926 (441) found that green manures retarded the disease and that superphosphates increased it. He continued the experiments with green manures and superphosphates until 1931 and by that time was able to say conclusively that green manures retarded the disease but where the superphosphates were added the disease was increased.

Bose (77) found that arhar after tobacco was less attacked by *Fusarium vasinfectum* than when following other crops. Pigeon pea was more vigorous after tobacco than after some of the other crops. This may have been due to the freedom from disease.

Selection among the resistant varieties offers hope. Plyman 612 reported that in the C. P., Strain No. 3 gave the highest yield of grain. McRae (445) stated in 1932 that he had found resistance to the disease in the A-2 strain of arhar. Shaw (679) crossed Pusa Type 5 and No. 8. the former susceptible and the latter resistant, and found a ratio of 9:7 or 27:37 with resistance being dominant.

Because of the wide host range of the fungus rotation does not offer much hope. It would seem that the best hope we have is the organic manures.

Diplodia Disease of Pigeon Pea

Host plants. It has been reported on pigeon pea by Raychaudhuri but so far has not been recorded as serious.

Geographical distribution. In India it has been reported only in Pusa.

Appearance on the host plant.

A thickening of the region of the collar with more or less distortion are the first symptoms to be observed. Small eliptical lesions develop along the margins of the distortions which later develop into definite cankers. If these are deep the stem may break and the plant fall. If callous tissue forms fast enough the plant may survive. The stems often become twisted as a result of the unequal growth of the wood tissue. Discoloration may appear a few inches above the cankers.

Adventitious roots may develop above the cankers which also are an aid in maintaining the life of the plant. As a result of the fungus invasion the tissues become a slate blue in colour.

The organism. Diplodia cajani Raychaudhuri Sp. nov.

The mycelium is septate, hyaline at first later changing to olive green and then brown to black when viewed in mass. The pycnidia are simple, globular and at first beneath the surface, later emerging. Their size varies from 300 to 475 microns.

The conidia are borne on short, slender conidiophores, they are like the mycelium, hyaline at first later turning a light brown. They are two celled, ovoid to elliptical, many of them being egg shaped According to Raychaudhuri the average size of those measured at Pusa 25.1 × 12.7 with a range of 21.5 to 30.1 × 10.8 to 12.9

Control. No definite control measures have been suggested at this time. However in line with the control measures suggested for other diseases of this sort it would appear that building up the soil fertility, rotation of crops and better soil drainage should help in reducing infection.

Cercospora Leaf Spot of Cajanus.

Hosts. The only host so far reported is *Cajanus indicus.*

Geographic distribution. Reported from the Allahabad and Pusa areas.

Appearance on the host plant. The symptoms first appear on the under side of the leaves small light brown spots 1 to 2 mm. in diameter. Roundish at first they later become angular and may coalesce to from large irregular blotches. The mid rib usually acts as a barrier.

Lesions may appear on the petioles and stems. Conidiophores are light brown in colour when young but become darker when older. Old conidial scars leave geniculations on the conidiophores. The conidiophores may branch. The size and septation number varies with the moisture content of the air.

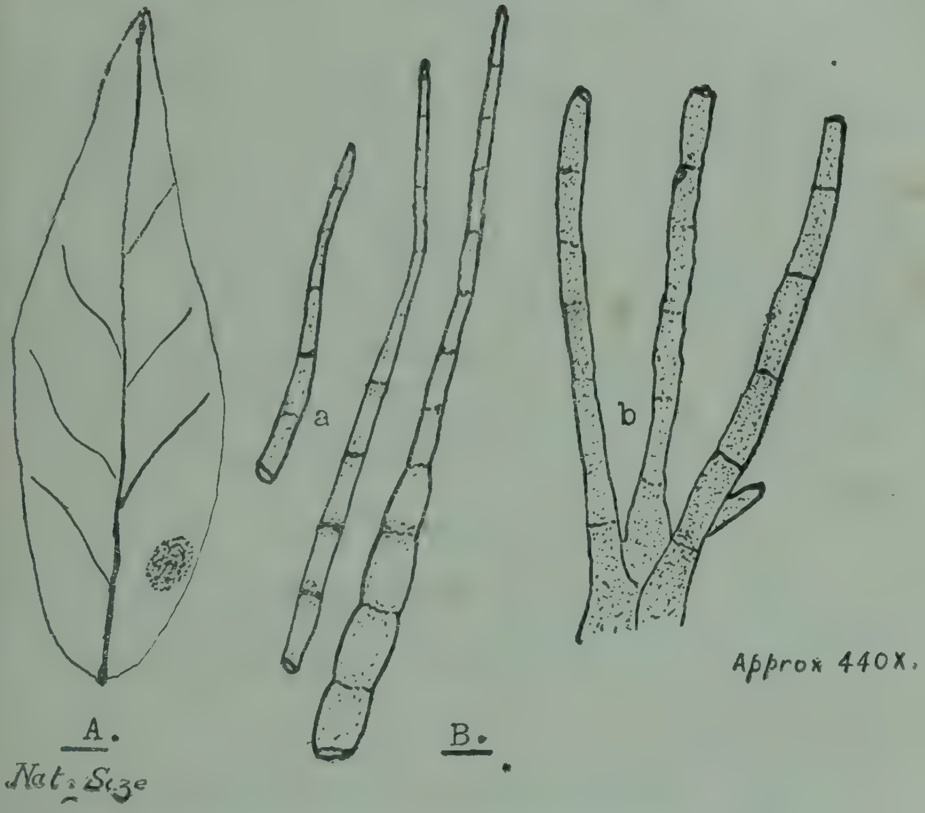


Diagram of *Cercospora indica* Singh on arhar.
A. Infected leaf.
B. a. Conidia b. Conidiophores.

The organism. *Cercospora indica* Singh.

The mycelium is both inter-and intra-cellular. Conidia are hyaline to slightly greenish. There are constrictions near the septations. Size varies from 68-129 × 3.4-5.1 microns. Septations from 0-9 with the mode as 2.

Conidiophores range from 28.0-168 ×3.4-7.0 microns with from 2-13 septations.

Control. At the present time there has been no serious epiphytotic of the disease so that aside from rotation, no control measure has been worked out.

The More Common Diseases of Gram in Northern India.

Gram is grown widely in the northern part of India and is one of the very important crops. Aiyer (9) states that it is grown on over 15,000,000 acres of which some 12,000,000 are in the northern part of the country. Gram wilt is one of the most serious. Gram blight, caused by *Micosphaerella pinodes*, is serious in the North-West Frontier. Gram rust does considerable damage in some sections during the 1946-47 season. *Sclerotium sclerotium* is also common on gram in some sections. Another gram blight is also sometimes serious. This is caused by *Phyllosticta rabiei.*

Fusarium Wilt of Gram

Hosts: Gram and possibly other closely related legumes but the host range appears to be limited.

Geographic distribution. Northern India and possibly Burma.

Appearance on the host plant. Among the first symptoms is a distinct drooping of the leaves following by a wilting and then necrosis of the tissues of the collar and mainroots. When diseased plants are pulled up the lateral roots are likely to break and remain in the soil. These are usually badly diseased and discoloured. Diseased plants are likely to be scattered over the field rather than in continuous areas.

The organism. Padwick in 1940 (572) determined that the organism responsible for gram wilt should be called *Fusarium orthoceras* Appel and Wollenweber var. *ciceri.* Appel and Wollenweber set up the section *Elegans* among the *Fusarium* on the basis of no sporodochia, no pseudopionnotes or sclerotia. The species *orthoceras* has white mycelium and forms a wine-red culture when rice media is used. According to Sherbakoff (690) the microconidia are in greater numbers than the macroconidia. The macroconidia measure 25-40×3.2-4 microns.

Padwick stated that the fungus could live over in the roots and stems of the gram.

Control. As in the case of other soil borne fungi organic manures are of value in the control of the fungus. Padwick and Bhagwagar (583) determined that late sowing was also a factor in the control. They found that October sowing was much better than September. Of the dates tried October 14 and October 21 were the best dates. In 1941-42 the wilt on the September sown plots was 64.5 per cent. That on the October 14th sown plots was 10.8 per cent and that on the October 21st. was 5.0 per cent. At a later date the same author stated that the wilt appeared related to drouth.

Some evidence has been secured in favour of resistant varieties but the most practical effect at this time appears to be organic manures.

Gram Blight

Hosts. Peas, beans, vetch, gram and other legumes.

Geographic distribution. World wide.

Appearance on the host plant. Butler (93) mentions it as causing heavy losses in the North-West Frontier as early as 1911. He states that the symptoms are very similar to those of anthracnose on bean. The infected spots appear on the leaves first and then on the pods. The spots tend to assume the shape of the structure on which they occur. That is on the leaf they are round and on the stems they are elongated in the direction of the long axis of the stem. As the spots mature they become brown on the margins with a yellow-gray center which is dotted with the pycnidia. This is known as the *Ascochyta* stage. At one time it appears to have been confused with a *Phyllosticta* but *Phyllosticta* is a single celled pycnospore-forming fungus where as *Ascochyta* possesses a septum making it a two celled spore.

The spots progress on the pods rapidly and the seeds are infected or even prevented from forming. Luthra and Bedi (392) record data of weighing seed from infected and normal pods and found the weight of the two lots of 100 seeds each to be 3.95 and 12.57 grams respectively.

The organism. Microsphaerella pinodes (B. and Blox.) as the perfect stage and *Ascochyta pisi* Lib. as the pycnidial stage.

The perthecia are numerous over the infected areas. The members of the genus *Mycosphaerella* produce their perithecia only after the death of the host plant. The asci are typically 8-spored, the spores hyaline to greenish, ellipsoid and 2-celled. Perithecia vary from 100 to 140 microns, varying with the conditions under which they grow. Pycnidia are black, composed of angular cells. Pycnospores are hyaline, more or less pink in mass, and measure from 9-20 × 3-6 microns. Perithecia are round, brownish in colour, measure from 100-300 microns. Ascospores are two-celled and slightly constricted at the septum.

Control: Sattar (668) stated that the disease was worst in the Punjab when the rainfall was over 6″ for the period October to April. In the Punjab when such a condition occurred the infection was over 50 per cent but when the rainfall was 3.5″ the per cent of infection dropped to 25 per cent. Susceptibility of the plants appeared to increase with age of the plant. The plants were more susceptible at the flowering and fruiting stage than any other time.

The disease is seed transmitted but, while both internally and externally transmitted, the spores on the outside of the seeds were the more important source of infection. For the control of these seed borne spores

Sattar (668) recommends clean seed and seed treatment.

For the internally borne seed spores he suggests soaking in water at 20° C for 6 hours then in water at 55° C for 15 minutes. For the externally borne spores he suggests a 0.5 per cent copper sulphate solution for 10 minutes. Destroy all refuse and rotate. Sattar (668) considers that *A. pisi* and *P. rabiei* are the same fungus.

Luthra and Bedi (392) reported in 1932 that gram blight caused by *Phyllostica rabiei* was serious in the Punjab. They reported that all aerial parts of the plants were infected. It had a temperature range of 15-25 which would make it a cool temperature fungus. Sattar's report was made two years later and it appears that they were dealing with the same fungus.

Control. In 1938 there was a severe epiphytotic in the Attock Dist. of the Punjab. This in spite of the usual precautions which had previously been effective. On that year the weather took a hand and wind borne spores were the source of new infections and thus the disease spread. As a result of the failure of local control measures, varietial trials were made and one variety, F_8 was found that gave great promise. Luthra et.al (400) reported that in 1940-41, 40,000 acres of this new selection were sown in the Punjab. This large acreage was made possible by the effort of the cooperative agencies in increasing the F_8 as rapidly as possible to replace the susceptible varieties.

Macrophromina Rot of Gram

Hosts: Wide range.

Geographic distribution. World wide.

Appearance on the host plant. The disease appears on the mature host plants as a bronzing of the leaves on the lower branches. The colour changes to yellow and then brown. The diseased branches are rigid and stiff and likely to curve upwards. The leaflets take various angles but more likely to be verticle and soon fall. The roots are brown to black and after a time become shriveled. Dastur (158) states that sclerotia were not found on the plants but they were readily formed in pure culture from the isolations. He states that the Macrophomina disease was the most serious on gram in the C. P. in 1935.

The organism. Macrophomina phaseoli (Maubl.) Ashby. (*Sclerotium bataticola Taub.*)

See under potato and cotton.

Control. Rotation and use of organic manures.

Dastur (158) in the C. P. tested a number of varieties from different parts of India and found that the Poona and Cawnpore varieties were most resistant.

Gram Rust

Host. Appears confined to gram *Cicer arietinum.*

Geographic distribution. It has been recorded in most of the countries where gram is grown.

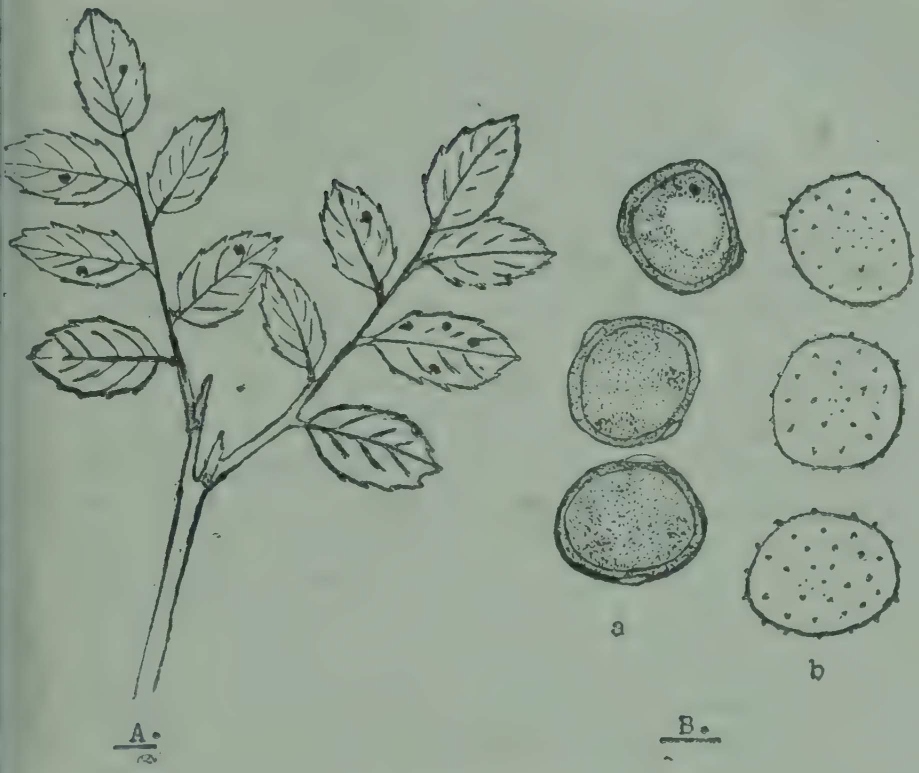
Appearance on the host plant. On the leaves it appears as small oval brown powdery pustules which may coalesce to form larger ones. While occurring on both surfaces, they are more common on the lower.

The organism. Uromyces ciceris-arietini (Grogn.) Jacz.

The uredospores are round brownish-yellow with minute spines on walls. They measure 20×28 microns, often contain as many as 8 germ pores.

Telutospores are similar to the uredospores but more variable in colour. They are round to ovate with a thickened apex and a warty, roughened wall. They possess but a single germ pore.

Control. No control measures have been suggested except rotation of crops. It is not usually serious but the year 1946-47 was unusual in that the gram crop was very seriously hurt in parts of the Punjab and the C. P.



Diagrams illustrating Gram rust (*Uromyces ciceris-arietini*).
A. Type of infection.
B. Uredospores a. Cross section. b. In outline.

Sclerotinia Sclerotiorum (Lib.) Mass.

This species are formerly known as S. *liberatiana Fel.* but, according to Wakefield (914), should be called S. *sclerotiorum* (Lib.) Massee.

Host plant. As mentioned above, it has a wide range of host plants, many of which are of economic importance and widely cultivated in India. Up to the present time, however, it may be considered among the minor diseases on these crops. Among the host plants of economic importance in India are; *Brassica campestris* var. *sarson; Avena sativa; Cannabis sativa; Cicer arietinum; Hordeum vulgare; Lathyrus sativus; Linum usitatissimum; Pisum sativum; Triticum vulgare; Vicia hirsuta; Zea maize* and others.

Geographic distribution. The fungus appears to be widely distributed in many countries.

Appearance on the host plant. The symptoms are variable and appear to be subject to the influence of the environment in which the host plant is growing. On the leaves the infections appear as small brown areas on both sides; they may spread to involve the entire leaflet. If the leaflets are heavily infected they may fall to the ground and then develop the heavy saprophytic infection which is also a stage of the fungus. A crown rot may also be evident in fields of alfalfa, clover, peas and vetch. Mundkur (509) reports a boll disease of *Hibiscus sabdariffa* due to a *Sclerotinia* which he identified as S. *sclerotiorum*.

The organism. Sclerotinia sclerotiorum (Lib.) Massee. Although the asexual stage of the fungus has been reported to be a *Botrytis*, most of the workers are of the opinion that it has not been definitely shown to be so and consequently should not be considered such at this time.

The white mycelium is found on and beneath, the surface of the host plant, specially where there is much moisture, as in the axils of the leaves, between bud scales, in bulbs and at the surface of the soil. For that reason it has been found to be a serious rot of bulbs in some cases.

The mycelium is much branched, septate and rich in protoplasm. On the outside of the host tissue it acts as an infection agent when it comes in contact with a portion of the plant which is healthy.

When the food supply is exhausted and the vegetative growth has ceased, the mycelium becomes very dense in spots and within these spots are formed the typical sclerotia. At first these are pink and later they turn black and become smooth. They form on leaves and within the tissues of such plants as carrot and lettuce.

The sclerotia can germinate at once or after a resting period. In some cases they will remain dormant for several years. Mundkur (509) found that the sclerotia were incapable of causing infection unless they were cut, as in the case of the *Sclerotinia sclerotiorum* which he isolated from *Hibiscus*. That is, they could not cause infection without producing apothecia and ascospores. After they were injured they were able to produce infection in contact with the host tissue just as the mycelium does.

When the sclerotia germinate they send up small sprouts which can attain the length of some 5 centimeters. In the light, or upon the surface of the sprout being exposed to air, the sclerotium thickens and becomes flattened, developing into an apothecium which is cup shaped. The asci are cylindric, 130-135×8-10 microns. The spores are ellipsoid, 9-13 × 4-6.5 microns and minutely guttulate (with tiny drops). The ascospores and mycelium are both short lived and the fungus can travel but a short distance in the soil.

Control. In many cases the use of sprays does not seem practical for the control of *Sclerotinia* diseases. In the case of the one on roselle, Mundkur (509) recommends hand picking of the sclerotia and deep plowing. Bauer and Huber (48) found the use of calcium cyanamid in a dust form to be effective in protecting against S. *fructicola* on blossoms of stone fruits. It is possible that it may be equally effective on such crops as lettuce. But for the field crops it has not been considered economical to use sprays.

At this time it would appear that the best control would be rotation of crops and the destruction of sclerotia together with deep plowing and resistant varieties.

The More Common Diseases of Ground Nut in Northern India.

The ground nut is subject to the attack of some serious fungus species. Root rotting fungi, such as

Sclerotium rolfsii and *Macrophomina phaseoli* are serious in the Allahabad area. *Cercospora personata* is serious when the weather is optimum for the fungus.

Cercospora Leaf Spot of Groundnut

Host plant. The groundnut *Archis hypogaea L.*

Geographic distribution. It is common in Africa, the United States, Australia, South America and Asia, including the Philippines.

Appearance on the host plant. The disease appears first as small dark brown circular spots on the leaves when they are from one to two months old. They are often surrounded by a bright yellow ring. The size varies from a mere speck to one third of an inch in diameter and there may be as many as a dozen

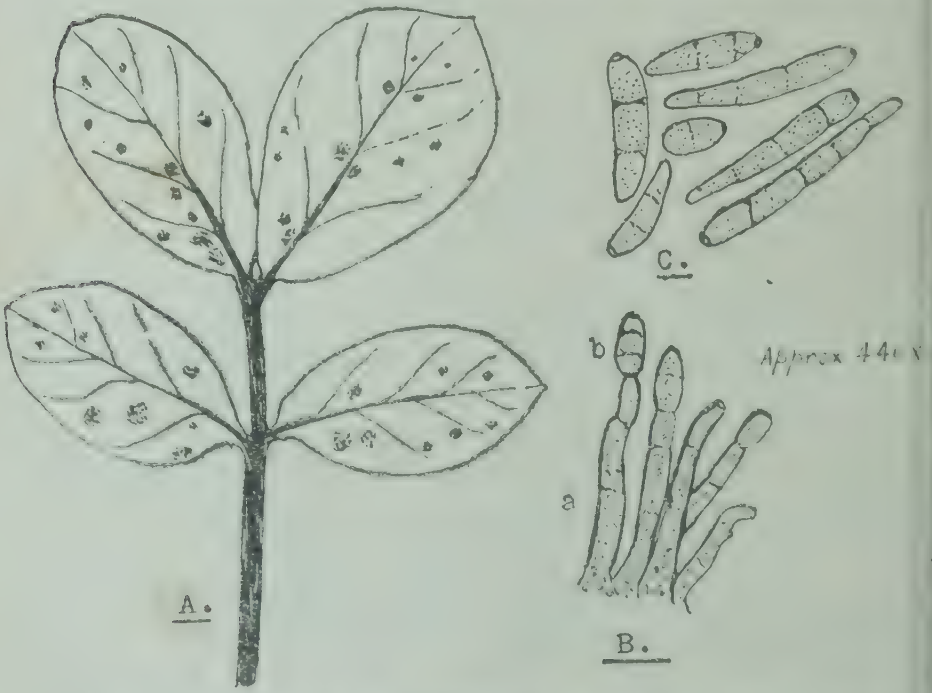


Diagram illustrating the appearance of Tikka disease (*Cercospora personata*) on groundnut leaf.

A. Infected leaves.
B. a. Conidiophores.
C. Conidia enlarged.

on a single leaf. When attacks are severe, loss of the leaves is nearly certain to follow. In fact, falling of the leaves may follow a light spotting. When this occurs only the bare stems remain.

The organism. Cercospora personata (B & C) Ellis.

Jenkins (Journ. Ag. Res. LVI pp. 317-332, 1938) placed the fungus in *Mycosphaerella berkeliyii* as the perfect stage has been found.

The mycelium is found confined to the spots in the leaf and does not appear in the green portion. It seems that the fungus kills the plant tissue immediately so that it limits its own growth and progress. As the tissue dies the mycelium collects in masses under the surface and from these masses numerous spiral threads are sent up against the epidermis and rupture it, or they emerge through the stomata as conidiophores. These appear in clusters and the clusters may be in more or less concentric rings. The conidiophores are short, uniseptate, possess the knee joints (conidial scars) and are of the typical olive green colour. Conidia are short for *cercospora*, being from 20-55 × 6-8 microns and possess from 2-7 septa. They are irregularly cylindrical, the longer ones being slightly curved.

It is believed that it is soil borne as well as disseminated by the wind. There is no evidence that it is seed borne.

Control. Sanitation and rotation appear to be important control measures. Miller (480) reported that sulphur (325 mesh) proved best for the control of the leaf spot. By the use of sulphur dust treatment the average yield of treated over untreated in Miller's experiments was 235 per cent.

The Georgia Experiment Station Report for 1945 gave results of the use of sulphur and copper and sulphur as dusts for the *Cercospora* leaf spot which produced 438 pounds more nuts to the acre than the untreated.

Sclerotium rolfsii Sacc.

Host plants. The fungus has a wide host range having been reported on a number of economic plants in India. Butler and Bisby (96) record it on *Solanum tuberosum, Arachis hqpogaea, Piper betle, Delphinium spp., Medicago sativa, Cicer arietinum, Lens esculenta, Triticum valgare, Eleusine coracana* and *Sesbania grandiflora.* In America it causes blight of wheat and many other grasses and is serious on sugar beets. In India it has been reported on apple, on mango, on cotton and other plants.

Geographic distribution. World wide.

Appearance on the host plant. The infected plants lose their colour, wither and turn brown. The disease occurs in patches and these are marked by a general retarding of the growth of the plants.

The organism. *Sclerotium rolfsii* Sacc.

The perfect stage is known as *Corticium rolfsii* Curzi.

The mycelium is white, coarser than that of *Rhizoctonia*, with small brown, spherical sclerotia occurring on rotted roots, corms and culms. The fungus is very aggressive under favourable conditions of moisture and can be both saprophytic and parasitic, producing a dense white cotton like mass of mycelium over diseased or decayed parts.

The sclerotia form as tiny mustard seed like bodies and are produced in large quantities.

The fungus grows best on wet soils and such soils are usually unhealthy for the plants. The continuous growing of certain crops, such as root crops, etc., on the same soil will cause increases in the disease.

In the Philippines (555) *S. rolfsii* was found attacking young cotton seedling and causing a snapping of the stems of older plants. Inoculations into lettuce, soy beans, tomatoes and rice were successful.

Mundkur (510) produced the perfect stage on onion, asparagin proteose peptone media. Galloway also reported the production of basidia and basidiospores by the same method. In 1937 Narasimhan (539) also recorded the production of basidiospores, thus establishing the fact that the perfect stage was not difficult to secure and that it probably occurs in nature.

Control: Crop rotation and sanitation help in reducing the disease but as the sclerotia remain viable for some time such measures do not completely control it. In Mysore (539) the use of 1% Bordeaux has proven helpful in controlling the disease on apple roots. The use of mercury dusts for control has been suggested together with resistant varieties in the case of ground nuts.

Chamberlain (101) states that if potatoes are rotated with *Brassica* spp. and legumes the amount of infection is reduced. He found that after four years in grass the disease was greatly reduced.

Leach and Davey (367) used aquous ammonia (50 p. p. m.) for 24 hours exposure and found that it was toxic to the sclerotia. In field trials in California they found that ammonium sulphate at the rate of 300 p.p.m., dissolved in irrigation water was sufficiently toxic to reduce the root rot of sugar beets. Bertus (58) believes that the use of decayed vegetable matter containing the fungus, for purposes of mulching was possibly the means of introducing the disease into young grafts in Ceylon.

When ginger rhizomes were found infected in storage with S. *rolfsii,* a 1-1200 solution of mercuric chloride for 1½ hours at (a) two months and (b) three months, gave 687 and 728 healthy plants in 1,000 as compared to 470 for the untreated plants.

From the above it would seem that the control of the sclerotial diseases among the various crops is not easy and that each crop presents a different problem and requires different treatment.

Root Rot of Groundnuts.

Hosts: Extremely wide range.

Geographic distribution. World-wide.

Appearance on the host plant. The plants are likely to become yellow and sickly looking. The symptoms of this organism (*Macrophomina phaseoli*) may be confused with those of *Sclerotium rolfsii.* In fact it is probable that isolations may have to be carried out before the real identity of the fungus can be determined.

The organism. Sclerotium bataticola Taub. (*Rhizoctonia bataticola* (Taub.) Butl.

Macrophomina phaseoli (Maubl.) Ashby.

McRae (449) reported the finding of the pycnidial stage of the fungus on ground nut. See root rot of cotton. The pycnospores are 10-18 × 5-6 microns.

Control. Resistant varieties appears the chief hope at the present time. Thomas (798) writing from Madras stated that trials with a large number of varieties of ground nuts there was some resistance shown by some of the varieties tried. A. H. 45 was the most resistant of the varieties tried at that time (1941). In Madras it was found, just as in cotton, that March and April sown ground nuts contracted more of the disease than those sown in May. The percentages of infection were 38.9, 35.8 and 4 respectively.

Hopkins (292) dusted the shelled nuts with 5 oz. of mercury compound per bag of nuts (180 pounds) and was able to control the root rots. The Georgia Experiment Station Report for 1945 gave a report of the control of root rot of ground nut with 2 per cent Ceresan, 2 per cent Spergon and 2 per cent Arasan.

Some Common Diseases of the Bean in Northern India.

The garden bean is subject to a number of diseases. Perhaps the most serious would be anthracnose. Rust may at times become serious but not usually. Cercospora leaf spot may also be serious. Root rotting fungi will also do serious damage at times. Virus disease causes some damage.

Bean Anthracnose.

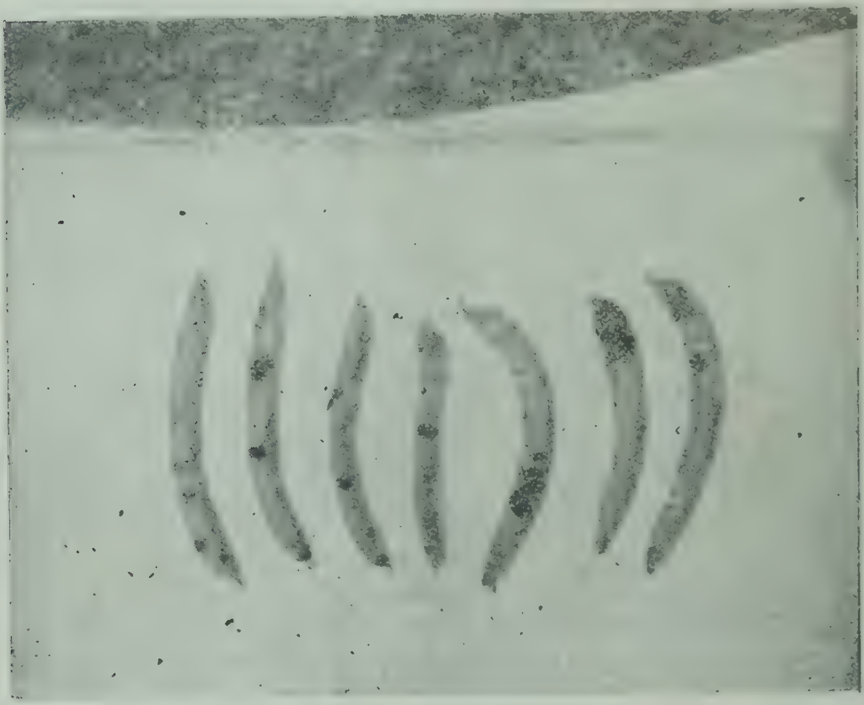
Host plant. Confined largely to varieties of the common bean (*Phaseolus vulgaris* L.), bean anthracnose has however been reported on several other host plants, including the scarlet runner (*P. multiflorus* Willd.) and the lima bean (*P. lunatus*). Barrus (Cornell Memoir 42, 1921-recorded it on *Citrulus vulgaris.* This is doubtful and may have been due to the confusion between the anthracnose of bean and melon. He also recorded it on cowpeas (*Vigna sinensis* Endl.) Butler (93) also reported the fungus on cowpea in India. In addition he reported it on val (*Dolichos Lablab* L.) and on horse gram (*D. biflorus* L.).

Geographic distribution. World wide.

Appearance on the host plant. The fungus may attack any portion of the bean plant. The most prominent symptoms are on the pods where it first appears as tiny brown specks in epidermis. As these enlarge they become dark in the center with coloured borders. They vary in size from tiny specks to one centimeter or more and where two or more unite they may involve the entire pod. The colour of the pod may produce some effect upon the spot type. Spore masses on the lesions are flesh coloured at first and later drying to a brown or black granular mass. Setae characterize the acervuli but a hand lens is required to see them.

On the leaf the diseased areas are on the under side. especially on the veins. Only a small part of the leaf is usually killed but if the petiole or large veins are attacked the whole leaf may die.

Seedling may show infections on the cotyledons and these areas may become covered with the flesh coloured spore masses. Seeds are infected from the pod and show discoloured spots. These may extend through the cotyledon into the embryo. Roots do not appear to be affected.



Photograph of bean pods infected with anthracnose, (Colletotrichum lindemuthianum). Photograph taken at Shillong, Assam.

The organism. *Colletotrichum lindemuthianum* (Sacc. & Magn.) Bri. & Cav. A fungus very similar to the bean anthracnose was isolated from beans and in culture produced asci and ascospores and was accordingly named *Glomerella lindemuthianum* Shear. Later work with proven cultures of *C. lindemuthianum* failed to repeat the findings and for the present it would be best to retain the use of the imperfect fungus name.

The mycelium is localized in the lesions and does not spread. Acervuli are organized beneath the tissues and soon rupture through. The conidiophores are simple, measuring from 45 to 55 microns. The conidia are single celled, oblong to ovoid, straight or slightly curved and average 15 by 5 microns. For a more complete discussion of the fungus see Heald (275) pages 679 to 691).

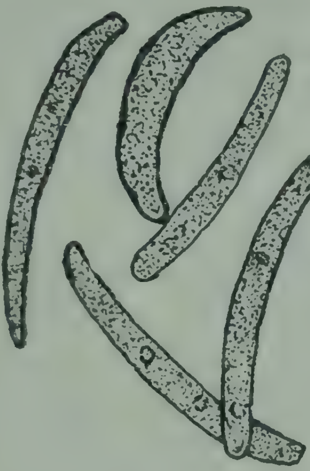


Diagram of conidia of bean anthracnose, *Colletotrichum lindemuthianum*, grown on oat meal agar.

Control. Rotation and destruction of diseased host material will help. Resistant varieties are one of the most dependable control measures. It is not found in the U. P. as a serious disease but is much more common in the hilly regions of the C. P.

The Bean Rust

Host plants. The bean rust has been reported on species of *Phaseolus, Dolichos* and other related legumes. It has been reported in India on mung, urd, cowpeas, French beans and others.

Geographic distribution. The rust is widely distributed over the world wherever the host is grown.

Appearance on the host plant. The rust is usually found on the leaves although the stems and pods may also be attacked. The sori are small, nearly round and a powdery brown in appearance. They may be scattered or coalesce to form larger areas. The damage is mostly to the leaves.

The organism. Uromyces appendiculatus (Pers.) Fries. As previously mentioned, the rust possesses all four spore stages. The aecidial stage is similar to that of the next rust to be discussed (*U. fabae*), occurring frequently but mostly on the under side of the leaves. The aecidiospores are somewhat oblong in shape and from 18-36 by 16-24 microns in size.

The uredospores are 24-33 by 16-29 microns and bear numerous minute spines. The teleutospores are 26-35 by 20-26 microns, smooth, dark brown and possess small, hyaline wart like papillae at the tip. The sori usually appear on the older plants late in the season and develop beneath the epidermis. Teleutosori follow the uredosori and are either on the under side of the leaves or on the stems. They are smaller than the uredosori and appear as black wart-like structures surrounded by a yellow halo like band. The teleutospores are unicellular and capable of remaining viable for some time and germinating the following season. The basidiospores are capable of infecting the bean plants and thus initiate the infection.

Life cycle. This begins with the early basidiospore infections and passes through the aecidiospore, uredospore and teleutospore stages on the same host plant.

Control. Although sulphur has been shown to be effective in the reduction of the rust infection, at the same time it has not been shown to be practical in the field. Early harvesting and immediate destruction of the vines is probably the best measure to use as that prevents the teleutospores from infecting the next season's crop.

Bean Leaf Spot

Host plants. Various legumes such as Dolichos, mung, and French bean.

Geographic distribution. Wide spread wherever the legumes are grown.

Appearance on the host plant. The spots begin as brown or red areas with gray centers and purplish borders. They may be rounded but are usually angular because of the vein limitation to spread. The centers often break away leaving holes. In 1939 and again in 1941 mung plants on the Institute farm were completely defoliated by the fungus.

The organism. Cercospora cruenta Sacc.

The conidiophores emerge in clusters through the stomata as brown, septate hypha which possess the knee joints marking the conidial scars. They measure from 50 to 11 by 4 to 6 microns. Conidia are variable as in the case of most *Cercospora*, being from 50 to 100 by 4 to 5 microns and 2 to 8 septate. When grown in a moist chamber on mung leaves the conidia often reach a length of 175 to 200 microns with as many as 14 septa.

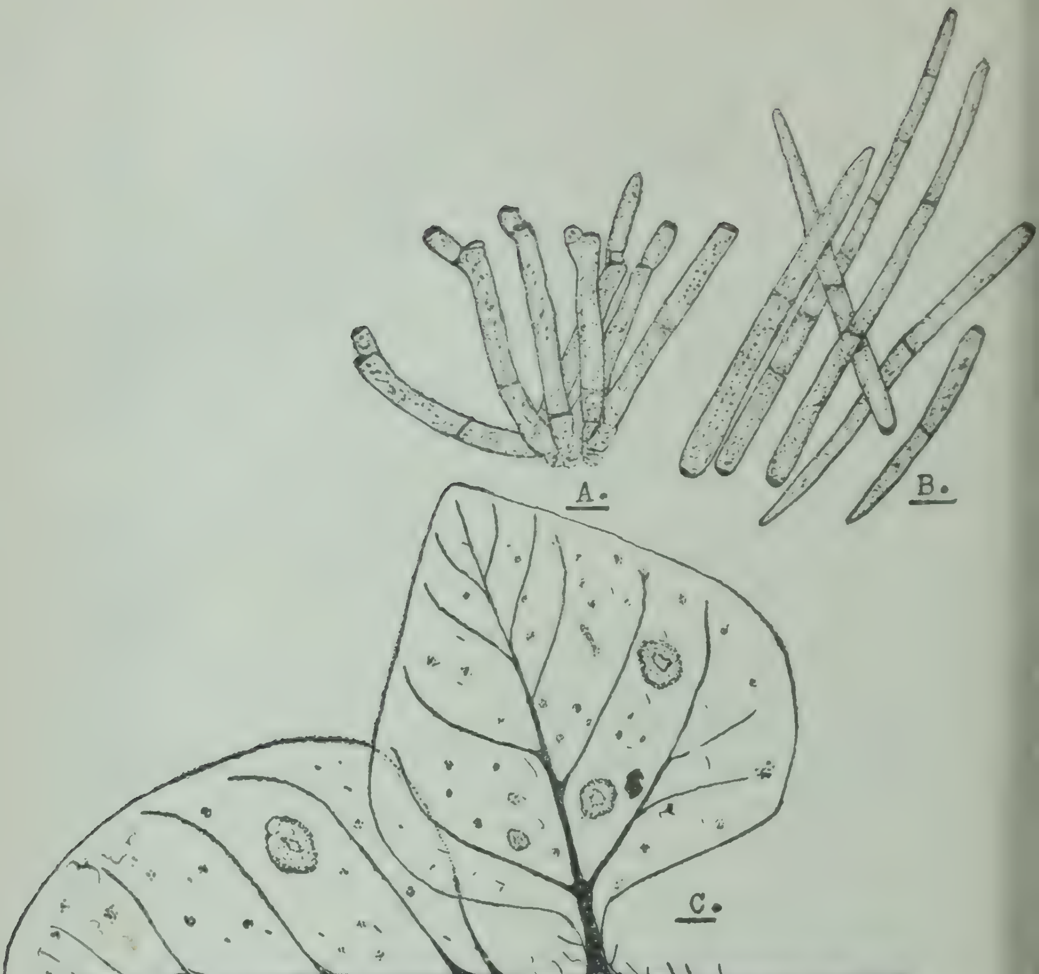
The first leaf spot was observed on mung in the Institute plots on August 17 in 1941. This was on P. 101 and on August 24 spots were visible on P. 58. By October 1, all of the varieties were infected in varying degrees from complete defoliation of P. 58 to scattered spots on P. 23. The five varieties being grown in the plots exhibited the following order in susceptibility to the leaf spot; P. 58, P. 101, P. 35, Local and P. 23.

Control. Sanitation and rotation are the only measures at this time.

Root Rotting Fungi of the Bean

The diseases of the bean roots consist of rotting and wilting. Wilted plants will usually have a number of the roots destroyed by fungi but some of the wilting may be due to stoppage of the vascular system.

Hosts: The fungi which have been found on the bean are also found on most of the other field crops, especially the legume crops.



Diagrams illustrating the Cercospora leaf spot of mung.

A. Conidiophores.
B. Conidia.
C. Infected leaf.

Geographic distribution. World wide.

Appearance on the host plant. Among the first symptoms will be the yellowing of the leaves. This yellowing will not be confused with that of the yellow bean moasic as in the case of the root rotting the whole leaf becomes uniformly yellow. Leaves droop in the day time and may revive at night. Blossoms are likely to be few and they may abort. Examination of the stem at the surface of the ground will usually show dark areas that extend down to the small roots. If the plant is removed from the soil the roots will be found dark and many of them rotted and broken. Often it is possible to see the mycelium of some of the fungi on the outside of the main roots. The center of the stem at the surface of the ground will be dark and on examination will be seen to have fungi in the vascular tissue. Sclerotia may be seen on the outside of the roots and stem.

The organism. In this case as is many others, there are more than one organism that cause the root rotting. Among the more common will be;

- *Rhizoctonia solani,* Kuhn.
- *Macrophomina phaseoli,* (Maubl.) Ashby.
- *Fusarium species.* (Probably *F. vasinfectum* Atk).

Control. Soil borne organisms are difficult where-ever met. Rotation of crops does little good unless the choice of crop is made with care as the fungi are extremely cosmopolitan and have a wide range of hosts. Green manures and barnyard manure are an aid. Good drainage during the time when the plants are small is a distinct help.

Virus Diseases of Bean (Phaseolus vulgaris)

There is little Indian literature on the virus diseases of the common bean, *Phaseolus vulgaris.* There is at least one virus on the beans in the vicinity of Allahabad. This is the yellow bean mosaic Virus or *Phaseolus Virus* 2 Pierce. Others may exist but they have apparently not been thoroughly worked out.

The yellow bean mosaic is characterized by a faint mottling in the beginning which consists of numerous dark green areas with small light yellow spots developing in the green background. The yellowing gradually increases so that the leaflets appear chlorotic. The larger leaves become somewhat curved upwards and may appear glossy in appearance. The mottling becomes more pronounced as the season advances. The plants become stunted and there is a tendency to become bushy.

The virus is able to infect the garden pea, white clover, the soy bean, the lupines, and other legumes. Although it appears to be common on the garden bean it has not been seen on the garden pea. Perhaps it is here on the pea but observations have been incomplete.

The Common Disease of the Pea in Northern India

The garden pea is subject to the attack of some fungi which become serious when conditions are favourable for them. The downy and powdery mildews are both serious under some conditions. Rust (*Uromyces fabae*) is also serious some times.

The Pea Rust

Host plants. The rust is found on peas, broad beans, lentils, sweet peas and many species of *Lathyrus* and *Vicia.*

Geographic distribution. Common in many countries. It has been reported on all of the above hosts in India.

Appearance on the host plant. The conspicuous stage of the rust is the uredo stage. The sori are on both sides of the leaves and often appear in circles. The sori are light brown to golden in colour. When infection is heavy the leaves may become deformed and wither early.

The organism. Uromyces fabae (Pers.) de Bary. The first appearance of the rust is the aecidial stage which occurs on the under side of the leaves but may occur on other parts of the plant as well. Spermagonia (pycnia) will also be found mixed with aecia. The aeciospores are elliptical to rounded with fine wartlike spines on the surface, more or less yellowish in colour and measuring from 14 to 22 microns in diameter. The uredospores are light brown in colour, possess spines and measure 20-30 by 18-26 microns with from 3 to 4 germ pores.

The teleutospores are more likely to be found on the stems. They are darker in colour than the uredospores and this difference is reflected in the colour of the sori. The teleutospores possess thick walls, are subglobose, ovate or elliptical with a rounded or flattened apex. They measure 25-38 by 18-27 microns. They possess a pedicel which is once to twice the length of the spore remaining attached to it when mature. They germinate to form a four celled promycelium with basidiospores produced in the same manner as in the cereal rusts. These initiate the early infections.

Hiratsuka (282), after a study of the species of U. *fabae* in Japan, was convinced that at least three physiologic forms existed there.

Life cycle. The life cycle follows the order of the spore appearance since they all appear on the same host in the same season.

Control. At present the same methods for control hold for *Uromyces fabae* as for *U. appendiculatus.* Rotation of crops should, however, be added to those control measures already mentioned.

Powdery Mildew

Hosts. It has one of the widest host ranges of any parasite. It has been reported on nearly three hundred host plants many of which are of economic importance. The fact that it's name indicates a relationship to the *Polygonaceae* is of little value as an indication of the host relationship since the fungus is one of the most destructive parasites known on legumes, especially on the garden and field peas. It has often been of much more importance than the downy mildews.

Geographic distribution. Its distribution is world wide as it has been reported from every country in which the legumes, upon which it is parasitic, are grown. In many sections of the world the crops are seriously damaged. In the northern part of the United States the clovers are attacked and the crop may be severally damaged.

Appearance on the host plant. The disease makes its most serious attack during the time the plants are fruiting. About the time of flowering a white powdery material will be seen on the leaves and stems of the plants while some of the leaves will show hypertrophy and distortion. The areas of powdery material will vary in size, in extreme cases completely covering the plant. During dry seasons there is more effect than during wet. As the plant ages the colour may become more or less grayish brown and the leaves and stems have a dirty appearance.

The organism. The commonly accepted name of the fungus is *Erysiphe polygoni* D.C. The mycelium is thin, effused and web like, often disappearing. The perithecia are in clusters or scattered and usually smaller than the perithecia of other genera. The appendages are of variable number, interwoven with the mycelium, hypha like and more or less flexuous. The asci are usually two to eight, but may be more, are of variable shape and size and may or may not, be stalked. The ascus size varies from 46-72×30-45 microns with from 3-8 spores measuring from 19-25×9-14 microns. The perithecia persist in the soil on old leaves until the following growing season of the plants they parasitize and the ascospores are scattered at the proper time in the host development.

Conidiophores are multicellular and the conidia are borne in chains. They are ovate and hyaline. They germinate immediately and are the main agent of spread of the disease during the growing season of the host plants. As the conidia germinate they produce an appresorium at the tip and from this a haustorium is quickly developed which is sent into the underlying tissues. These are smaller than most of the *Erysiphe* species and somewhat less lobed.

Physiologic specialization has been shown by Mains (413) to occur on clovers, and by Blumer (69) on *Pisum*, *Lathyrus*, and *Trifolium*.

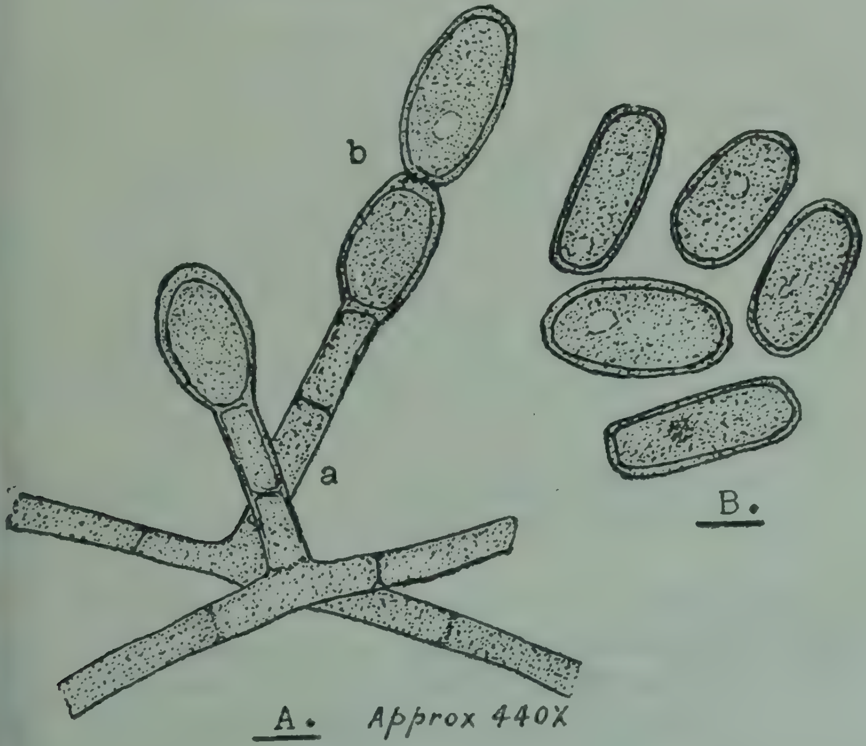


Diagram of conidiophores and conidia of *Erysiphe poylgoni* causing powdery mildew of pea.

A. a. conidiophore b. conidia.

B. Conidia enlarged.

Life cycle. The life cycle begins with the time the host plant is about ready to blossom and is initiated by the ascospores which have been liberated from the perithecia. These spores fall upon the host plant and soon start an invasion of the surface tissues. Soon conidia are produced and the disease spreads rapidly. The close of the season brings the production of perithecia and these carry the fungus over the long rest period until the host plants are in the proper stage of growth again.

Control. The use of finely divided sulphur is the most effective means of control for the mildew. Uppel (849) reported complete control of the mildew on the pea in the Bombay Presidency by the use of 20 lbs. per acre of finely divided sulphur at flowering time. But the use of sulphur on a commercial scale is hardly practical for the average farmer and resistant varieties seem the best solution to his problem.

The Downy Mildew of Field and Garden Pea

Hosts. The garden pea (*Pisum sativum L.*) and the field pea (*Pisum arvenae L.*) are the principal hosts. There are other hosts which are attacked. It has been reported (96) on *Vicia hirsute*, *Lathyrus sativus* and *Trogonella polycerata.* It has been reported on lentils in the Punjab. In most cases the damage is slight.

Geographical distribution. It has a wide geographical distribution. Probably as wide as that of the host plant.

Appearance on the host plant. It appears on the leaves of the field and garden pea, forming a downy growth on the under surface of the leaves, the spots varying in size. These may cover the whole under side of the leaf or only portions. When fruiting is abundent the spots assume a grayish violet colour and upon examination the spots are seen to be covered with many conidiophores. These are typical of the *Peronospora* conidiophores. Infected leaves soon become slightly deformed and withered, later falling from the plant.

The organism. Peronospora viciae (Berk.). de Bary.

The mycelium is composed of typical coenocytic hyphae which are intercellular, except for the somewhat branched haustoria which penetrate the host sells. Conidiophores arise from the intercellular mycelium directly and emerge through the stomata. They are some what longer, before branching, than the conidiophores of *parasitica* but the branching is the same and the conidia are similar. The conidiophores measure some 400-700×9-11 microns. The branches are slightly less curved than those of *P. parasitica* and are a pale violet in mass. Conidia measure 22-27×15-19 microns.

Oospores are round, light brown in colour and the epispore is raised with larger reticulations. They measure from 28-32 microns. The oogonium is thin walled and after fertilization the oospore fits tightly into the envelope. This is the resting spore and the means of survival through the dry periods.

Life cycle. The life cycle is simple. As the young peas emerge from the soil, the oospores germinate and infection tubes penetrate the young leaves as they emerge. As the disease progresses conidia and conidiophores are formed and the fungus is spread from plant to plant by wind, water and other agencies. With the formation of the oospores at the end of the season the life cycle is complete.

Control. As the disease is not of major importance in India effective control measures have not been worked out. In some cases cutting the tops before oospore formation has prevented the disease from being carried over the dormant period of the crop plant on which it is parasitic.

CHAPTER XI

SOME COMMON DISEASES OF LINSEED, CRUCIFERS AND CUCURBITS OF NORTHERN INDIA

SOME OF THE COMMON DISEASES OF LINSEED IN NORTH INDIA

The linseed is one of the important crops of many parts of India. It is often grown in between other crops, such as barley, gram etc. Among the more important diseases of lindeed are rust (*Melampsora lini*) Wilt and blight caused by *Alternaria lini.*

The Linseed Rust

Hosts. The linseed rust appears to be confined to the cultivated species of *Linum*, although there are biologic forms which occur on wild species, such as *L. rigidum* Pursch.

Geographic distribution. It appears to be world wide wherever the host plant is grown.

Appearance on the host plant. It is an easy rust to identify because of the bright orange coloured uredosori which are conspicuous on the above ground parts of the plant. Later the sori turn dark and then black as the teleutospores develop. It is autecious, all of the spores forms are found on the same host plant. The aecia which are found on the lower surface of the leaves are some 200 to 400 microns and a bright orange in colour.

The organism. Melampsora lini (Pers.) Lev. The rust has been given a number of names at one time or another which include the genera *Uredo, Podo-*

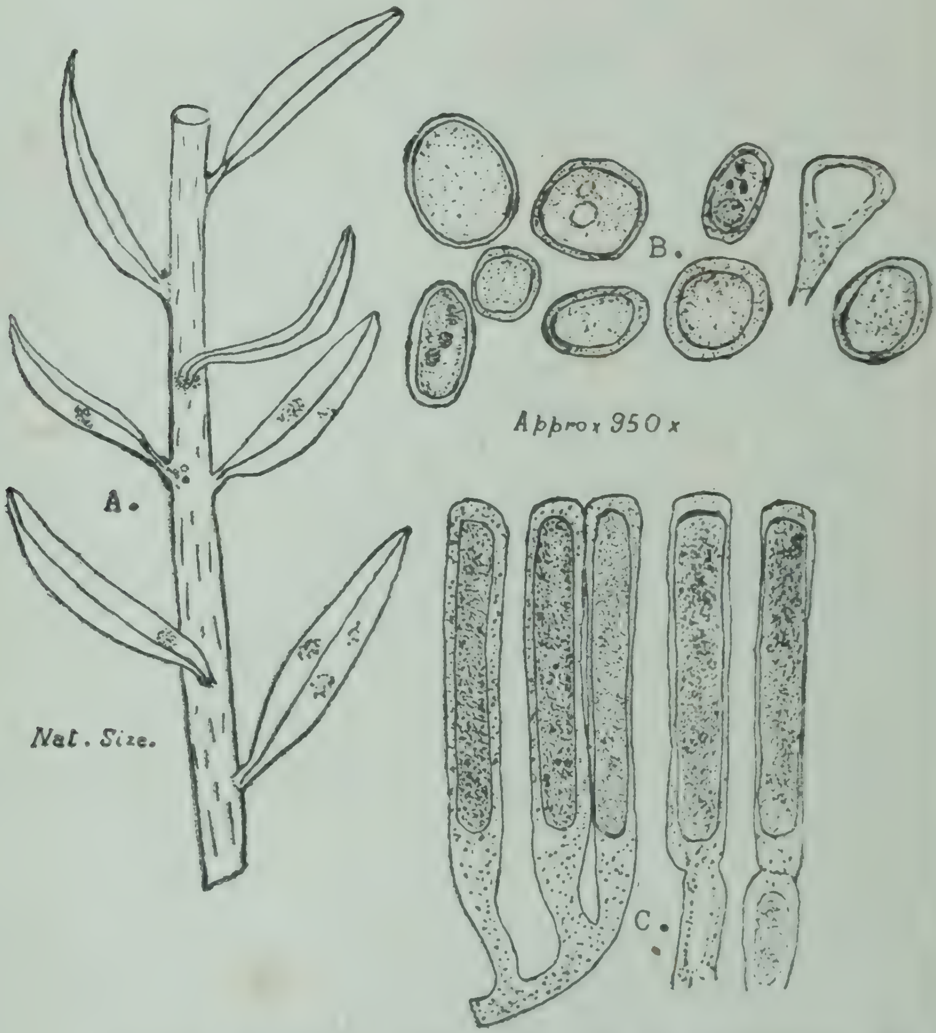
sporium, *Lecythea* and *Xyloma* as well as others. As previously mentioned, the rust is autecious and considered as a long cycle rust because all of the spore forms are found on the same host plant.

The spermagonia are found on the young plants and are small, inconspicuous, pale yellow, flattened, globoid or lens shaped bodies just beneath the epidermis. Aecida are mostly formed on the under surface of the leaf and the aecidiospores are formed in chains. They are rounded in shape, measure some 21-28 by 19-27 microns, and are warty, colourless walled bodies with no distinct pores. They are the product of a cell fusion in the base of the aecidium which results in a binucleate condition that persists until the teleutospore stage.

As the aecidia are on the common host plant, uredospores soon arise from the mycelium resulting from the aecidiospore infections. They appear on both sides of the leaves, on fruits and stems and occasionally on the floral parts. The uredospores are nearly round, with a colourless wall, orange coloured contents and measure 15-25 by 13-18 microns. Paraphyses are present.

The teleutospores, which follow the uredospores in the cycle of development are developed in reddish brown, later black, sori which often appear crust like on the stem. The spores are formed packed tightly together in a palisade like layer and average some 10 by 55 microns. Germination is typical of the rusts and in this case the sporidia are capable of infecting the economic host plant. This fact has no doubt been a factor in the wide distribution of the rusts over the world.

Miss Allen (21) determined the heterothallic nature of the linseed rust. She (20) was able to show that male gametes enter the stomata of the linseed leaf and fuse with the specialized cells of the hypha and that following such a fusion aecia would develop. She also determined that it was necessary to have both



Diagrams illustrating linseed rust caused by *Melampsora lini.*

A. Stem and leaves of linseed showing sori.
B. Uredospores.
C. Teleutospores.

types of mycelia present before the aecia would form. Unisexual (monosporidial) infections bore no aecia.

Physiologic races have been thought to exist for some time following the work of several investigators. Hart (267) found that the rust of common flax (*Linum usitatissimum*) could infect *L. rigidum* but not

L. lewisii while that from *L. lewisii* could not infect common flax. In 1940 Flor (229) concluded that at least 24 different races of linseed rust exist in the world. Of this number Padwick (567) believes that at least two are present in India. One of these is in the Pusa area and the other in the Karnal district.

Life cycle. As the life cycle is completed on the same host plant it is a simple matter to outline the steps from the formation of the basidiospores to the formation of the teleutospores at the end of the season. In sections where the winters are cold the teleutospores are the main link in the life cycle but in sections where the temperature does not go low in the dormant period it is considered likely that the uredospores are capable of acting alone to propagate the rust from season to season.

Control. Resistant varieties appear the best means of control. In 1925 Henry and Stakman (277) found that the variety Ottawa 770B was immune to the U. S. form and when Henry (277) crossed an Argentine flax with Bombay (C. I. 42) he found that the hybrid was immune. At that time he considered that resistance was dominant by a 15 to 1 ratio. In India, Plyman (613) has found E. B. 3 resistant to *M. lini* and that when E. B. 3 was crossed with local and some of the Punjab linseeds some very satisfactory hybrids were secured.

Sharville (Jour. Agr. Res. LIII pp. 81—127 1936) found that resistance to linseed rust was due to a number of factors. He was able to divide flax varieties into four classes according to resistance or susceptibility. There appears to be some morphological resistance which is connected with epidermal thickness and stomatal opening. Sharville also thought there might be some inhibitory influence exerted by the plant cytoplasm.

From the above it would appear that resistant varieties are the main hope of the farmer although rotation with other crops should aid in control.

Alternaria Blight of Linseed

Host plants. At present it appears to be confined to the linseed plants.

Geographic distribution. The United Provinces and northern India.

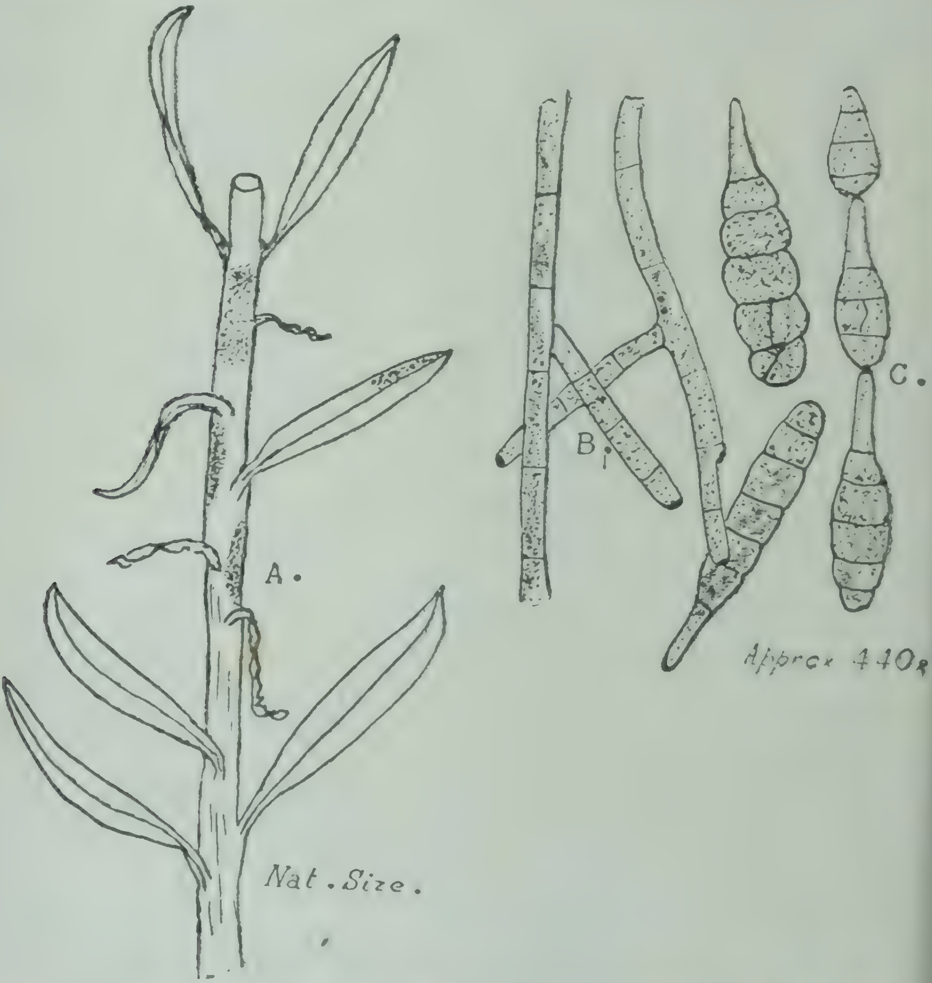


Diagram of stem of linseed showing infection caused by *Alternaria lini* Dey.

A. Stem and leaves with infection.
B. Conidiophore.
C. Conidia.

Appearance on the host plant. Minute dark spots appear at the base of the calyx. The flowers may not open or the floral parts may rot away. The fungus may spread into the stems and these may collapse. Dey (184) has given a complete description of the fungus on linseed.

The organism. Alternaria lini Dey.

Dey determined the spore size to be from 10 to 40 by 5 to 10 microns with a beak that measures 3 to 7 microns and possessing from 2 to 6 septa. The mycelium is nearly colourless with a faint greenish tinge which he called gray white in mass.

Control. The fungus has not become sufficiently widespread to be considered serious and no control measure have been devised.

Flax Wilt

Hosts: Linseed. But it is also a saprophyte and can live in the soil without a host plant.

Geographic distribution. Occurs in many parts of the world but was most serious in the northern part of the United States.

Appearance on the host plant. It may attack the linseed plant at any stage of its development. Seedlings may be killed before they produce the second leaves. Plants may be attacked and die at any stage of their existence. Being a vascular disease the vascular system is plugged and wilting, with yellowing of the leaves, is a major symptom. In the northern part of the United States the flax growers called it flax wilt and they said that the soil became "flax sick" and they soon learned not to plant flax on the same soil after the disease appeared. The truth about the disease was first demonstrated by Dr. H. L. Bolley, of the North Dakota Agricultural Experiment Station in 1901. Dr. Bolley was able to show that the cause of the sick soil was a fungus and at that time was able to offer little hope of control. The only thing open then was to seek new disease free soil and plant there. In a year or so move on to another field in which flax had never been planted.

The organism. Fusarium lini Bolley.

The spores are borne in masses (sporodochia) which are cream to flesh coloured. The conidiophores are branched, hyaline and short. The conidia are three-septate and slightly curved. They measure 27-38 × 3-3.5 microns.

Control. Resistant varieties are the only reliable means of control. Seed treatment is not reliable as the spores are borne on the surface of the seed but the mycelium is also within the seed coat and thus is readily carried on to new ground. In the United States the resistant varieties are Bison, Linota, Redwing and others. In India, Mc Rae (441) in 1926 reported that Pusa Type 121 and Sabour were highly resistant to the disease. In 1931 Alam (16) reported that in Orissa Sabour 6 showed high resistance to *F. lini* and that some good results had been obtained with hybrids between it and Pusa Type 12 and 121. It would seem that where the fungus is troublesome the resistant varieties may well prove the hope of the grower.

Root Rot of Flax

Hosts: A wide range of host plants.

Geographic distribution. World wide.

Appearance on the host plant. Although the root rotting may be associated with yellowing of the leaves and wilting, these characters are also those of the wilt and are not necessarily caused by the root rotting organisms. But examination of the roots will tell whether it is the vascular organism or a root rotting fungus. The roots will be diseased and decayed. Discoloration will not be confined to the vascular bundless as in the case of *Fusarium lini.*

The organisms. *Macrophomina phaseoli* (Maubl.) Ashby
Rhizoctonia solani Kuhn.
Sclerotium spp.

These fungi have been described under a number of other diseases and will not be treated in detail here. The farmer is not concerned with the species and description as much as with the control.

Control. *Macrophomina* has been known to be able to cause root rot of linseed since as early as 1930 when Sundararaman (763) reported it on that host in Coimbatore. In 1935 Uppal (849) stated that the fungus could cause the death of linseed plants at all stages of growth.

These fungi generally infect the plants growing under unfavourable conditions. Wet soil is one of the conditions favourable to the fungus and unfavourable to the host plants. Well drained soil is essential. Select resistant plants. Destroy old stubble where the soil is known to be infested.

COMMON DISEASES OF THE CUCURBITS IN NORTH INDIA

Cucurbits are among the most common of the garden vegetables for many parts of India. Fortunately not many diseases are found serious on the common varieties. Blossom blight, downy mildew, powdery mildew, mosaic and root rot are among the most serious.

Blossom Blight of Cucurbits

Hosts. It has been reported on a large number of host plants such as species of *Cucurbitaceae*, *Hibiscus*, including the rose of sharon (*H. syriacus*), the scarlet hibscus (*H. coccineus*), *okra* (*H. esculen-*

tus) and cotton, (*Gossypium herebaceum*). It has been reported on leaves of Cassia in wet weather. Dastur (Mem. Dept. Agri. Ins. XI pp. 129-144, 1921) found it causing a dieback of chillies in Bihar in 1921. Wiber (921) found the same thing in Florida and Su (752) reported it on chilies in Burma in 1935. Van Hall (866) reported on groundnuts in the Dutch East Indies.

Geographic distribution. From the above it would appear to be widely distributed.

Appearance on the host plant. The characteristic appearance of the fungus is a mass of the dirty brownish conidiophores which cover the affected portions of the host plant. At Allahabad it has been common on varieties of *Crotalaria* and brinjal and occasionally on tomatoes and chillies. In every case the infected portions could be identified from a distance of several feet by the mass of fungus. In the case of *Crotalaria* the attacked parts are killed back for a distance of one to two feet. The flowers, leaves and fruits of the brinjal plants are attacked. The fruits of the chillies and tomatoes are mostly attacked and the flowers of the cucurbits, especially the petals.

The organism. The organism most commonly associated with the disease known as blossom blight is *Choanephora cucurbitarum* (B and Rav.) Thax. Others may also be associated with the mould, especially on fruits but this is the most common one. The asexual reproductive stage is of the conidial type. The conidiophores are simple, erect with expanded tips which bear a number of cylindrical branches, each one of which forms a globular head from which the conidia are produced. These are mostly elliptical, bearing longitudinal striae and of a light to reddish brown colour.

Sporangia are rarely found in nature, being studied mostly in culture. They are globular structures, white, more or less pendent, becoming black at maturity. They vary in size from 35-100 microns. The spores are from 18-30 microns with smooth walls and may possess lateral or terminal tufts of hair like appendages. Like *Rhizopus* it is heterothallic. Zygospore formation is similar to that of *Rhizopus.*

The life cycle is paractically identical with that of black mold.

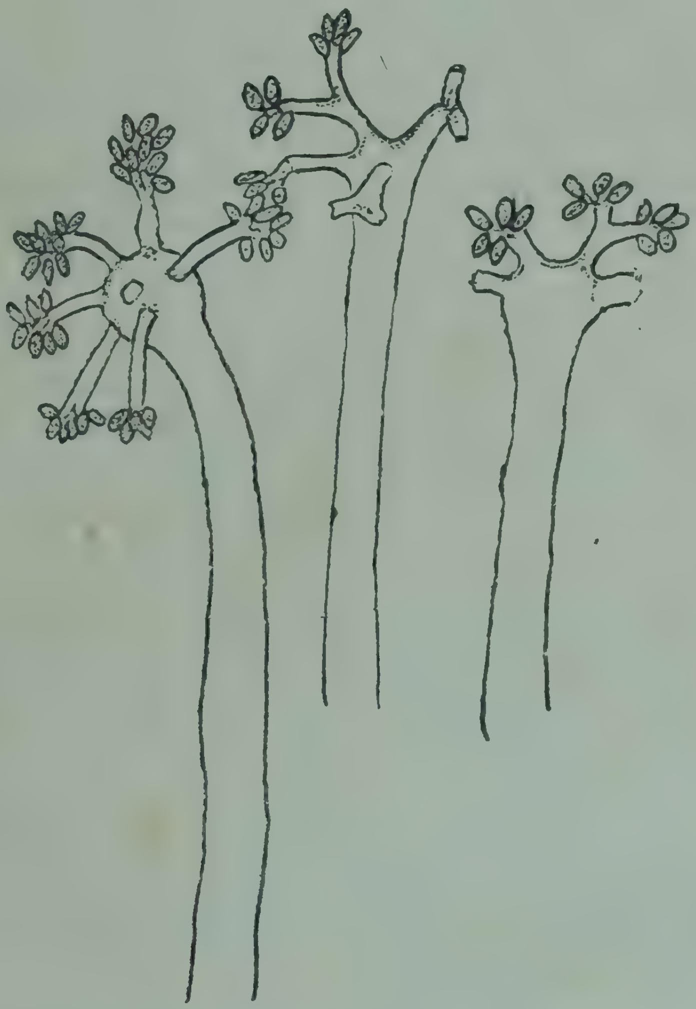


Diagram illustrating the conidiophores and conidia of *Choanephora* species. Diagram made from infected leaves of *Amaranthus* sp. growing in the Agricultural Institute Farm, Allahabad.

Control. In most cases control is not a problem but it is conceivable that under some circumstances it might become necessary. The fungus is spread by bees, the striped and spotted cucumber bettles, rain, wind and other agents. It would appear that a control programme would have to include an insecticide as well as a fungicide if it is to be effective.

The Downy Mildew of Cucurbits

Hosts. The fungus is parasitic on most of the economic members of the *Cucurbitaceae*, especially the gourds, melons and cucumbers.

Geographical distribution. Although known as an economic factor for only a comparatively short time, it is now found in nearly all parts of the world. First discovered in Cuba in 1868 on a wild plant it was soon discovered in the green houses of the United States and declared to have been taken there from Japan the previous year. Since then it has been found in many parts of the world and in some cases so severe as to be classed as a limiting factor in the growing of the Cucurbits in that section.

Appearance on the host plant. It is primarily a leaf disease, causing pale yellow spots to appear on the lower side. These spots may be angular at first becoming more or less irregularly rounded as they age. The colour deepens and becomes brown and with the spots limited on the sides by the veins they are easily identified. Infected leaves become deformed, dry and fall. Soft rot may develop in the base of the lamina or the petiole which some what resembles the late blight of potatoes but the leaf usually dies without any wet rot.

The organism: The organism *Peronoplasmopara* (*Pseudoperonospora*) *cubensis* (Berlese) Clinton, Was first called *Pseudoperonospora* by Rostowzew in 1903. but it is listed as *Pseudoperonospora cubensis* (B. & C.)

Rost. in many references. It produces a discoloration of the host tissue, producing a yellowing or water soaked appearance. The conidiophores arise from the stomata, usually 1 to 2. They are sparingly branched, the ultimate branchlets curving. Branch tips are acute and vary from 5 to 20. The conidia are gray, brown or smoky in colour, ovoid or ellipsoid in shape, possess papilla and measure from 20-40×14-25 microns. The oospores are spherical, yellowish-warty-papillate, 30-43 microns in size.

The mycelium invades the spongy mesophyll sending the conidiophores out through the stomata. Conidia germinate to form zoospores which are bicilliate, and flattened on the side of the cilia.

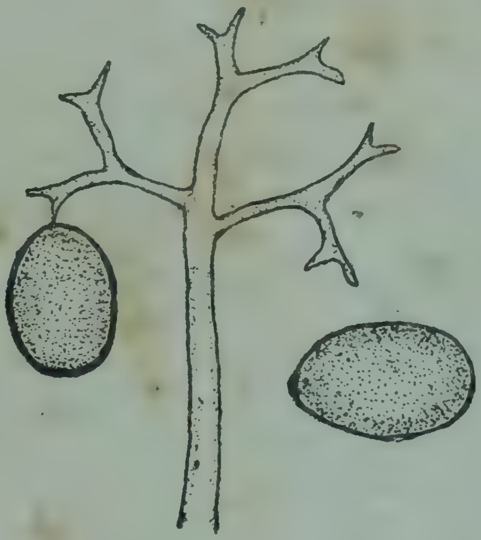


Diagram illustrating a conidiophore and conidia of *Pseudoperonospora cubensis.*

Moist weather is optimum for the fungus and sporulation is abundant during warm muggy days. The disease appears two to three days after infection with conidia appearing some ten days later. In some areas it is perennial. It may then spread annually into other sections. Oospores are rare. They have not been reported in most of the sections where the fungus is common and for this reason the ability to survive the year around is some what of a mystery. It must either live over on wild host plants which are unknown, or as resting spores which have not been found up to the present time.

Control: Where the fungus is severe Bordeaux has been found to control. In Illinois, U. S. A., Sayre (693) reports that copper lime dust composed of 16%, by weight, of dehydrated copper sulphate, 20% by weight, of calcium arsenate and 64%, by weight, of hydrated lime and used at the rate of 40 lbs an acre was effective in controlling the mildew. Butler (93) suggests a spray consisting of 2 lbs. copper sulphate, 2 lbs. lime and 50 gallons water as an effective control. This is Bordeaux mixture 2-2-50.

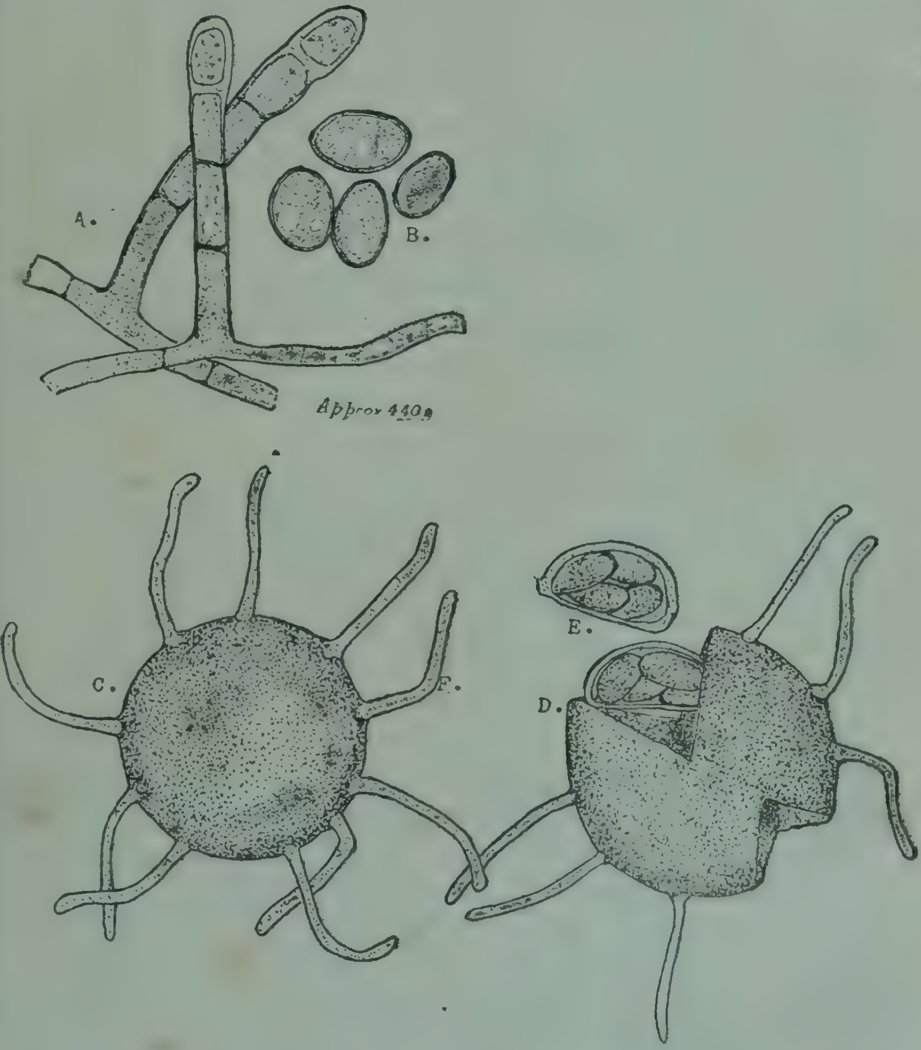
Powdery Mildew

Host plants. The above mildew has a wide range. of host plants, which includes many of the families of cultivated plants. Tobacco has been found to be attacked in South Africa, Thung (814) reported it in the Dutch East Indies. Fikry (224) reported it as one of the most destructive cucurbit diseases in Egypt, while Uppal (848) reported it as a destructive disease of cumin in the Bombay Presidency.

Geographic distribution. It is world wide.

Appearance on the host plant. It appears as a white powdery coating over the leaves and stems of the plants attacked. On okra it occurs mostly on the under leaf surface where it may cover all or most of the surface. The leaves lose their green appearance, may become pale dry up or even die. In the case of cucurbits, the leaves becomes distorted and the diseased areas are noticeable by their lighter colour. In the case of linseed, the leaves are covered with the grayish white powder and they become lighter in colour than the disease free leaves.

The organism. The organism is known as *Erysiphe cichoracearum* DC. Before the perfect stage



Diagrams illustrating structures of the fungus causing powdery mildew of Cucurbits.

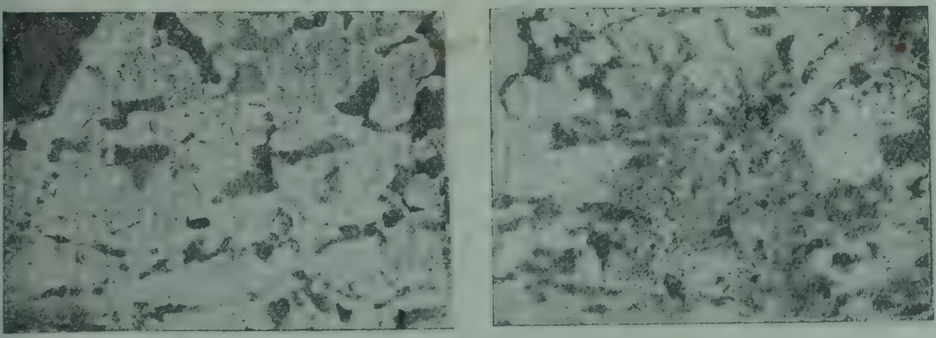
A. and B. Conidiophores and conidia of *Erysiphe cichoracearum* from pumpkin.
C and D. Ascocarps of the same fungus.
E. An ascus containing ascospores.

was found it was known as *Odium lini* Skoric. The mycelium is superficial except for the haustoria which penetrate the host cells and cause the reduction in activity of the plant parts. Conidiophores are short and bear chains of oblong rounded conidia which measure 24-30 × 15-20 microns.

The perithecia are not as common as in other species but they occur on many plants. On linseed they are produced on the dry or dead plant parts and are brown in colour. They are more or less scattered over the surface of the plant parts and measure from 80 to 140 microns. The asci are usually numerous (741) and measure 59-90 by 30-35 microns with a short stalk. Ascospores, usually two, measure 20-28 by 12-20 microns.

Life cycle. The life cycle of this mildew is practically identical with the one just described. Its vegetative stage parallels that of the host plants upon which it lives. It begins the season with the absorption of water and the expulsion of the ascospores. When these fall upon leaves of susceptible plants germination quickly follows with the production of haustoria, the further growth of the germination hyphae and the production of conidiophores and conidia. These are soon scattered to other host plants and the disease rapidly spreads from one part of a plant to another and from one host plant to another. With the maturity of the host plant the growth of the mildew ceases and the sex cells are then formed. Soon after this the perithecia appear and the season of activity is over for the fungus which depends upon the perithecia to carry it over the long dormant period. In some sections it is believed that the conidia are capable of passing over the rest period themselves.

Control. The use of sulphur has proven to be one of the most effective means of control of the powdery mildew. In the Dutch East Indies, D'Angremond (Proct-stat. Vorstenlandsche Tabak, Meded. 61 pp 1.131, 1928) secured control of *E. cichoracearum* on tobacco with 120 kg. of sulphur dusted on the soil per 0.7 hectare. If, however, the infection was heavy the sulphur failed to give control. Wager (904), who studied the shedding of mango flowers and fruits, found that Bordeaux 5-5-50 increased the fruit from 21 to 23%. In the Bombay Presidency (848) the use of sulphur dust, which will pass through a 200 mesh screen and applied with a Peerless Dust Gun, gave excellent control. Three applications were recommended at intervals of two weeks, the first soon after flowering. The total cost, including labour, for a tree twenty-five feet in height was 4 annas 9 pies and gave a profit of Rs. 7|0 to Rs. 8|0. Miller (480) reports that finely ground sulphur is the most effective means of control in the Imperial Valley of California. He reported that Bordeaux and dry copper dusts were not effective. When Corona copper carbonate, containing 18-50% copper, was applied, Guba found there was fair control and no damage to the plants, which in this case were cucumbers.



Photographs of pumpkin vines showing the effect of dusting with sulphur for the control of powdery mildew. The photograph on the left is of vines which had been dusted with finely divided sulphur, the one on the right is of undusted vines a few feet distant.

Mosaic Disease of Bottle Gourd

Hosts: Vasudeva, Sahar and Lal (878) have recorded the disease on cucumber, bitter gourd, melon, watermelon and vegetable marrow.

Geographic distribution. With the whole question of the virus disease symptoms in a state of uncertainty it is likely that the host range is much greater than this group.

Appearance on the host plant. Vasudeva et al (878) described the symptoms on the bottle gourd (*Lagenaria vulgaris* Ser.) as broad characteristic streaks between the major veins along which a deep green portion persists. This expands limiting the green portion to either side of the vein. Young leaves may be either distorted with wavy or irregular outline, may have a wrinkled surface or both.

The midrib may arch upward causing the leaf edges to turn down. Dark green blisters may appear over the leaf and cause wrinkling of the leaf. All succeeding leaves may develop the characteristic chlorotic streaks.

The organism. They did not name the organism except in terms of the disease symptoms and host. But from the symptoms described it is evident that the disease occurs on pumpkins as well for a virus with what appeared to be identical symptoms occurred on pumpkins at Allahabad in 1946-47.

Control. Control measures have not been given and it is largely theory on which suggested control measures may be based. At Allahabad the spread was apparently related to the increase of the sucking insects which were abundant on the vines.

Root Rot of Melons and Squash

Hosts: Butler and Bisby (96) list at least 18 hosts as being attacked by *Pythium aphanidermatum* in India and the host range in other countries is extremely wide.

Geographical distribution. World-wide.

Appearance on the host plant. The symptoms of the disease are wilting and rapid dying of the whole plant, where the disease is found under optimum condition for the fungus, with yellowing and more gradual dying under less favorable conditions for the parasite. The roots are soft and watery. A disagreeable odour is given off by the decaying tissues. On the fruit it may cause a blossom end rot which rapidly reduces the fruit to a watery mass. The growth is more rapid on melons than on squash fruits but root decay is similar in each case.

The organism. Pythium aphanidermatum (Edson) Fitz.

The organism has been described under the diseases of papaya. Yu (946) found that the optimum temperature for the organism to form sporangia was 24-26° C, with a humidity of 90 per cent. When the humidity dropped below 85 per cent the fungus was retarded. Gottlieb and Butler (245) found the optimum temperature in the southern part of the United States to be 37°C. They found that it would infect without wounds, apples, carrots, cantaloupes, cucumbers, egg plant, grapes, summer squash, sweet potatoes and tomatoes. At Allahabad the host plants from which it has been isolated are only papaya and squash. Papaya isolations have been mostly from the stems rather than roots. However, one isolation from the roots was from depth of 16 inches.

Yu (946) isolated the fungus from various types of soil and from a depth of at least 8 inches. He states that infection is always through the soil. Yu also says that the fungus can live over in the soil for four years and this adds to the problem of rotation for the farmer.

Control. It is evident from the above statement that there is need of a long rotation. On the average farm with the variety of crops which may be attacked by the fungus it is doubtful if any rotation will be satisfactory as a single control measure. Green manure and barnyard manure in the unrotted condition appear to offer some hope of control as the saprophytic fungi aid in holding the pathogens in check as well as giving the host plants a better start.

COMMON DISEASES OF THE CRUCIFERS IN NORTH INDIA

The garden crucifers are occasionally damaged by disease but there are relatively few that are serious. White rust, downy mildew, Alternaria and root rots are the most serious.

The White Rust of Crucifers

Hosts. It appears to be confined largely to members of the *Cruciferae* although other plants are also attacked. It has been reported on *Brassica*, *Capsella*, *Cleome*, *Gynandropsis*, *Nasturtium* and *Raphanus* by Butler and Bisby (96). It has been reported on horseradish (*Moringa oleifera*). On the Institute farm it is common on the tiger footed morning glory. (*Ipomoea pes-tigridis*).

Geographical distribution. It appears to be wide spread and to be found wherever the crucifers are grown.

Appearance on the host plant. The disease receives its name from the white powdery appearance of the spore masses and from the resemblance of the pustules to those of the cereal rusts. Infections take place on all above ground parts of the plant but are more common on the leaves, flowers and fruits. The infections first appear as slightly water soaked areas which soon become thickened and turn darker in colour. The plant part is likely to show slight distortion at the point of attack and if many infection areas are found on the same portion there may be distinct distortion which includes bending, curling of leaves, enlargement of fruit and flower and dwarfing of the whole plant.

The sori vary in size from a tiny speck to one half inch in diameter and from those that are depressed within the tissue to those that may extend out as much as one half inch from the epidermis. They are produced on the under surface mostly but appear on the upper surface as well. The upper surface will frequently show a depression, directly over the sorus, which is darker than the rest of the tissue.

The organism. The fungus is now known as *Albugo candida* (Pers.) Kuntze. Formerly it was known as *Cystopus candidus* Lev. Liro (391) reported the acceptance of the name *Albugo* for *Cystopus* and this would give the authority to use the above combination.

Infections begin with the swarm spore stage during the very early growth of the host plants and may occur on any part of the above ground portion, but is mostly confined to the parts mentioned above. It may occur by way of stomata or by direct penetration of the cuticle by the germ tube of the white rust spore. Once inside, the fungus spreads rapidly and soon infects a large portion of the host. At different points the fungus produces masses of mycelium which form a hymenium like layer and from which many upright hyphae are produced. These are the conidiophores and they cut of from the tip, by successive abscissions, the conidia. As the number of conidia increases the pressure upon the epidermis causes a rupture to take place and the exposure and distribution of the mass of white spores which bring about the spread of the disease. The conidia are able to germinate immediately and, if upon a suitable host, they cause further infections. Under suitable conditions of moisture and temperature the spread may approximate an epidemic.

Toward the end of the season in the older sori, antheridia and oogonia form. Copulation takes place and following fertilization the zygotes (oospores) are formed which are the typical resting spores referred to above.

The zygospores (oospores) will not germinate until some time after they have been formed. They are unable to escape from the host tissue until it decays which usually takes place during the rainy season. The zoospores escape, following the rupturing of the zygospore wall and infections follow. The fungus is a cool weather fungus and most of the infections are found during the cooler parts of the season.

A life cycle is easily constructed from the above description. Germinating zygospore liberate swarm spores which infect the host plants and result in the production of sori containing numerous conidia. These produce infections during the growing season of the host plant and at the end of the season the production of antheridia and oognonia result in the formation of zygospores which carry the fungus over the dry season.

Control. Rotation of crops appears to be the most generally accepted control. Copper containing compounds have been recommended for the control of white rust. Sanitation, clean cultivation to destroy wild host plants and the destruction of all refuse from the crop are also recommended.

The Downy Mildew of Crucifers

Hosts. This fungus is known to attack most of the crucifers in India (96), and has been found associated with other diseases in other countries. Ocfemia (555) found it associated with white rust on crucifers in the Philippines.

Geographical distribution. It is found wherever the crucifers are grown. In India it is wide spread and it is suggested that it may be able to infect other hosts than crucifers.

Appearance on the host plant. In general the appearance resembles that of the white rust *Albugo candida*, although there are no distinct pustules as in the case of *Albugo*. While there is a resemblance to *Albugo* injury, it may be said that the downy mildew causes most of its injury on the stem while the white rust is most serious on the leaves and flowers. Downy mildew may cause swelling of the stem for several inches with abrupt bending. On the flower the ovary and sepals may be greatly swollen and distorted. There may be a complete or partial deforming of the floral portion. When the attack is late in the season the portion affected may be small.

On the leaves there are lighter green areas visible on the upper side which are usually found to be covered with a thin grayish downy growth on the under surface. The downy growth is composed of conidiophores. In the case of very young plants the whole of the lower leaves may be attacked and become rotted. Occasionally the roots are attacked. This can be identified by the blackening of the tissues and a rot near the surface.

On mustard the deformed inflorescences may be seen for some distance in the field and they will be most likely to appear shortly before the wheat crop is ripe. Where mustard is grown in the wheat it is common to find the distorted flower parts appearing above the wheat.

If the deformed parts are examined in the early morning when there is a heavy dew or light rain, a grayish white cottony growth will be seen over the deformed surface. On cabbage and leafy crucifers there will be patches on the under side of the leaves.

Examination of this downy material will disclose it to be composed of many conidiophores and conidia.

The effect upon the tissues is to diminish the chlorophyll and increase the size of certain portions of the tissue. The cells of the parenchyma about the vascular bundles become enlarged, thus causing deformities.

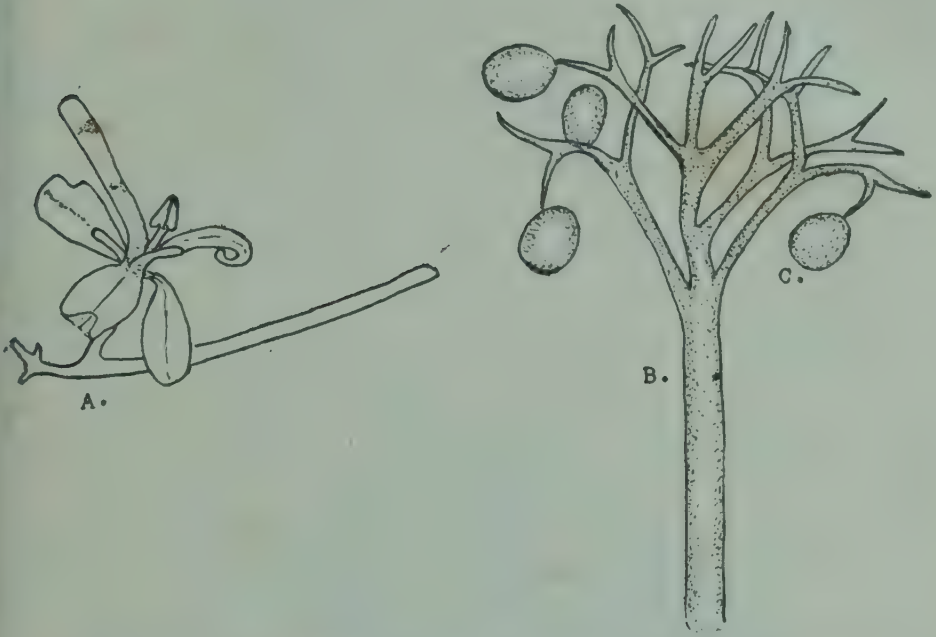
The organism. Peronospora parasitica (Pers.) Tul.

The mycelium of the downy mildew of crucifers is much like that of white rust in that it is intercellular. Haustoria are large, may be elongated, often much branched and may fill the entire cell.

Conidiophores emerge through the stomata and are erect, branched, often flattened at the point where they emerge from the stomatal opening, and may be twisted. They are 200-300 microns in length and bear conidia only at the tips of the branches. The branching is irregular, being more monopodial than dichotomous, each branch is repeatedly divided into two or several small branches which form acute angles and may show slight thickening just above the point of division.

Conidia are broadly ovate, blunt, often becoming globose, about 24-27 × 15-20 microns. They are colourless or nearly so. Oospores are borne in the interior of the host tissue and resemble those of Albugo. The oogonium is persistent on the oospore and is irregularly rounded and swollen into folds over the surface. The oogonium wall is pale yellow in contrast to the oospore which is yellowish brown and are from 26-45 microns in diameter.

Germination is by a germ tube in both condia and oospores. The germ tubes are not able to penetrate directly through the normal host tissue but enter by the stomata or where some injury has weakened the tissue. After the first infection has taken place, further infections are spread by the conidia which are formed in large numbers and scattered by wind, rain and other agents.



Diagrams illustrating downy mildew of Crucifers (Peronospora parasitica).

A. Mustard flower showing the distortion due to the infection by the fungus.
B. Conidiophore.
C. Conidium.

Life cycle. The fungus lives over the rest period in the form of oospores, although in Holland it has been recorded as over wintering in the old leaves (816). The first infections which result from the oospores are from the germ tubes of direct germination and take place soon after the plants are out of the soil and for the first few weeks are leaf invasions. As soon as the inflorescences appear these are also attacked. If weather conditions are optimum the spread may be rapid and an epidemic occur.

After the vegetative growth has passed its peak the oogonia and antheridia appear on the hyphae within the tissue. Soon the oospores are formed which are ready to carry the fungus over the following rest period. In India it is doubtful if there is much hibernating in the host tissue over the rest period.

Control. Venkatarayan (884) states that good control was secured in Mysore in 1937 by the use of Bordeaux mixture to which 1% groundnut oil had been added. Where bad epidemics occur the use of Bordeaux is recommended but usually the disease is not sufficient to make the use justified.

Photograph of a leaf of cabbage showing the early stages of leaf spot caused by *Alternaria* sp. (Note the arrows on the left which point to the spots.)

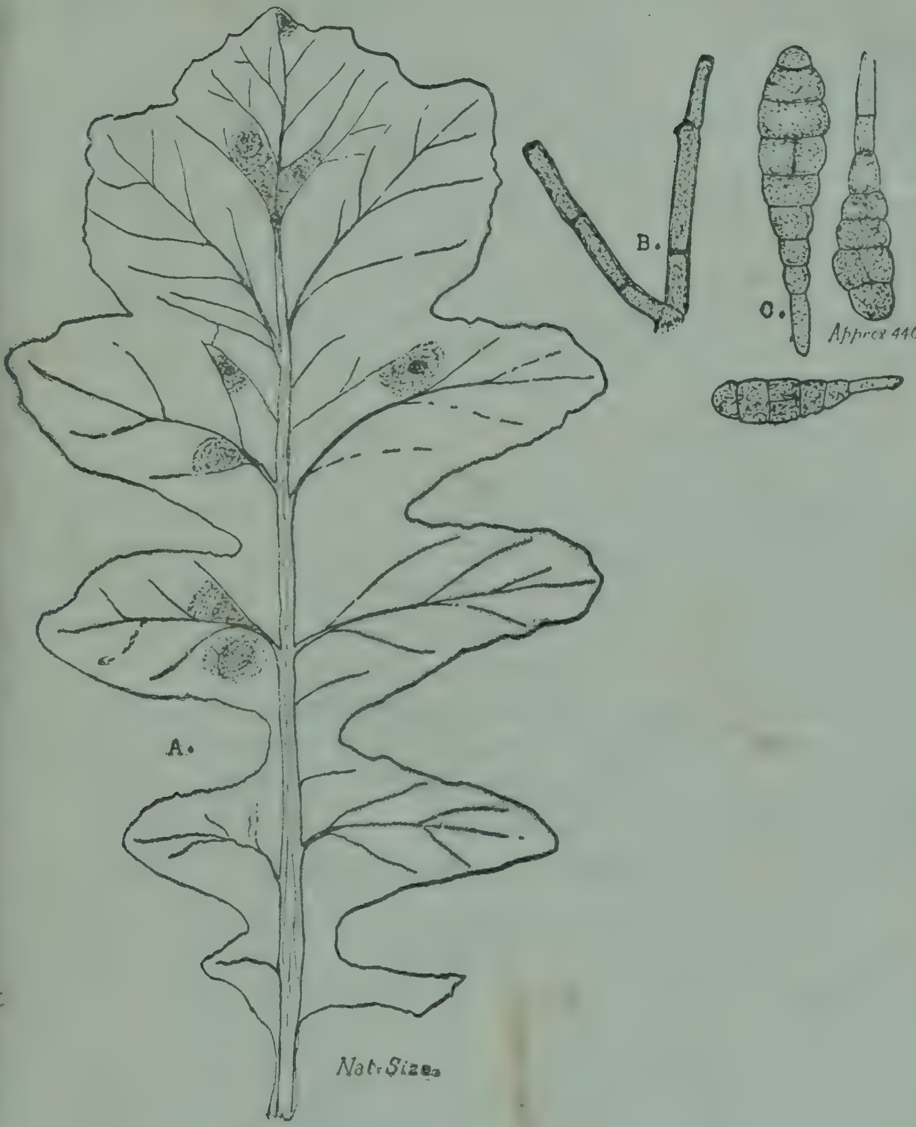


Diagram showing *Alternaria* on radish.

A. Infection on the leaf.
B. Conidiophore.
C. Conidia.

Alternaria Leaf Spot of Radish

Hosts: Radish, mustard, cabbage and other *Cruciferae*.

Geographical distribution. Apparently it is world-wide.

Appearance on the host plant. On the radish it forms concentric ringed, irregular spots which are dark toward the center where the conidia and conidiophores are mostly borne. On sarson it forms conspicuous, dark, more or less rounded spots, on pods and stem. It also occurs on the leaves but by the time the spots are apparent on the fruits and stems the leaves may have fallen. The conidiophores and conidia can be seen with a hand lens.

The organism. Alternaria brassicae (Berk.) Bolle.

Conidiophores are dark walled, short, with prominent cross walls and conidial scars. They are 0-4 septate with an average of 2. Conidia are somewhat elongate, fusoid (tapering toward one end) clavate (club shaped) and measure 60-80×14-18 microns with 5-9 septa.

Control. Rotation with non cruciferous crops. This is especially true in the case of sarson as it may become serious on that crop. It is usually of little importance on the other crops.

Root Rot Disease of Crucifers

In this case the Crucifers will be taken as a group instead of individually as the same root infecting fungi have been found on all of the *Cruciferae* in the Allahabad area.

Hosts: Wide spread.

Geographical distribution: World-wide.

Appearance on the host plants: This will be a general discussion of symptoms. Among the fungi commonly isolated from the roots of diseased plants in the Allahabad area have been species of *Rhizoctonia*, *Fusarium* and *Sclerotium*. The most common symptom of root infection in the case of cabbage or mustard is the yellowing of the leaves. Examination of these plants will show diseased roots and stem. Sometimes the stem above the ground line will show decay and in the case of *Rhizoctonia* or *Sclerotium* it may be possible to find the sclerotial bodies on the old dead roots.

Organisms. There are a number of fungi associated with the root disease of Crucifers.

Rhizoctonia solani Kuhn.
Fusarium species
Sclerotium rolfsii Sacc.
Pythium de baryanum Hesse.

Except for the species of *Fusarium* all of these have been described elsewhere. The *Fusarium* species have not been identified. *Pythium de baryanum* causes a damping off in the seed beds. Another *Pythium* was isolated from diseased cabbage roots on the Agricultural Institute Farm but was not identified.

Control: This is a major problem. Good drainage is an aid but rotation is difficult because of the wide host range of the fungi. At this time it is considered that the best control is the use of liberal amounts of barnyard manure that is put into the soil before being completely rotted and the rotting builds up the population of the fungi that are antagonistic to the pathogens.

Mustard Smut

A smut reported by McRae (442), which was considered to be *Urocystic corralloides* Rost., was found attacking the roots of *Brassica campestris* var, *sarson* near Pusa in 1928. Mundkur (516) found a smut on a species of *Brassica* in India which did not correspond closely to *Urocystis corralloides* and he concluded that it was a new species naming it *Urocystis brassicae.* The two smuts are apparently quite similar except that the spores of *U. brassicae* Mundkur are larger than those of *U. corralloides* Rost. The former can infect turnip, rape, cabbage, black mustard, (*Brasica nigra*) and *Brassica juncea.*

CHAPTER XII

SOME COMMON DISEASES FOUND ON THE FRUITS OF NORTHERN INDIA

Among the fruits which are included in the orchards of Northern India are the Citrus, the papaya, the mango, the apple, the peach, the grape, the pineapple, the jack fruit, the guava and many others. In this chapter some of the more common diseases of the orchard fruits will be discussed.

SOME COMMON DISEASES OF THE CITRUS FRUITS OF NORTHERN INDIA

The citrus fruits are a very important fruit crop in India. They are grown in nearly all parts of the country. Wherever a particular crop plant is grown in large acreages, it is certain to be followed by an increase in the diseases that attack it. The citrus fruits are subject to the attack of a number of serious diseases. Perhaps the most widely known and serious of the various diseases found on the *Citrus* species would be wither tip, canker, scab, gummosis and root rotting fungi. Of lesser importance would be some of the leaf spotting fungi and the fruit rotting fungi, such as the *Aspergillus* and *Penicillium* species.

Wither Tip of Citrus

Host plants. Although usually considered to be a *Citrus* disease it has also been reported on several other crops. Baker (42) reported the fungus causing a blossom blight and anthracnose of mango. Dey (186) states that two fungi are frequently associated with the wither tip disease. In Brazil it was reported on persimmons.

Geographical distribution. It is common in many regions of India although it is worst in the Punjab. It has also been recorded as serious in Cuba, the West Indies and Florida.

Appearance on the host plant. The most common character is the loss of leaves and the silvery gray colour of the twig tips. Acervuli appear on these diseased areas and in moist weather the pinkish spore masses may be seen with the naked eye. It is common to find setae around these acervuli. They are sometimes found on the diseased areas on the leaves but are mostly submerged.

The organism. Colletotrichum gloeosporiodes Penz. The perfect stage has been found to be *Glomerella cingulata* (Stonem) S. and V. S. (See ripe rot of chillies).

Gloeosporium limetticolum Claus. has also been found associated with the disease (186) and is sometimes differentiated from *C. gloeosporioides* by the absence of the setae but this is not a good character for setae are not always produced by the latter fungus. The acervuli of *C. gloeosporioides* range from 60 to 270 microns in diameter, are dark to black, the intensity of colour being proportional to the number of setae. The formation of the acervuli is the same as for the species described. The conidia are variable but according to Chaudhury (112) they average 13.2 by 5.5 microns. They are oblong, few slightly curved, mostly uninucleate.

The fungus grows best at a temperature of 25° to 27° C. and is found mostly along the foothills. In the United Provinces it does not develop much until the cooler weather of January and February and is not prominent until March.

Chaudhury (112) found evidence to support the idea that the fungus is heterothallic but perithecia were not produced. (See his article).

The fungus forms appresoria in the presence of moisture and some nutrient material. Dey (186) studied the formation of the appresoria of *Gloeosporium* and *Colletotrichum* and found them the same as described for *C. lindemuthianum.*

Under optimum conditions for the fungus. Chaudhury (112) found resistance only partial. He found Eureka lemon, khatti (rough lemon), turung and kimb (sour orange) giving evidence that they may be used for stocks and might have some influence on Malta scions. No resistant varieties of Malta were found.

Recently Sattar (670) reported the fungus serious on mangoes in the Punjab where it caused anthracnose similar to that reported by Baker (42). He found it on leaves, twigs and fruits causing numerous oval brown spots. The optimum temperature for the fungus was 25° to 29° C. which corresponds to that determined for the fungus on *Citrus* by Chaudhury.

Control. Dey (186) recommends removal of all diseased twigs and coating the cut surfaces with white lead formalin solution. Spraying with Bordeaux was also recommended, the formula being as follows:

Fresh stone lime	5 lbs
Copper sulphate	1½ lbs

Slake the lime in 5 gallons water. Dissolve the copper sulphate in 5 gallons water and mix. Make up to 25 gallons. It may be necessary to spray every three months, once before the blossoms open, and twice during the post blossoming period. See Fawcett (213) for a discussion of the disease in the U. S.

Canker of Citrus

Host plants. This disease is confined largely to the genus Citrus although it has been reported on species of *Atalantia, Fortunella, Feronia, Microcitrus, Poncirus* and *Xanthoxylum* (96). The lime, (*Citrus aurantifolia*) is one of the most susceptible but the grape fruit (*Citrus paradisi*) and the lemon (*Citrus lemon*) are also susceptible. Elliott (207) lists the pummelo (*C. grandis*), the sour orange (*C. aurantium*), the common citron (*C. medica*), the King orange (*C. nobilis*) and the trifoliate orange (*Poncirus trifoliata*) as susceptible, at least under artificial inoculation. Fawcett (213), however, lists them as resistant. He evidently means that they are not what would be termed as susceptible under natural conditions.

Geographical distribution. It has been reported in most of the citrus growing sections of the world although in some portions of the United States, as well as Australia and South Africa, the disease has been eradicated. It is wide spread in India, which is probably due to the extensive production of the kaghzi nimboo (*C. aurantifolia*), one of the most susceptible species to canker.

Appearance on the host plant. The bacteria attack all parts of the growing portion of the tree, even including the thorns. On the leaves, the disease appears as small yellowish brown spots which become slightly raised in the middle and then erupt. They usually appear on the lower surface first. As they grow older the tissues break in the centre of the spots and become rough and warty in appearance. They may increase to one fourth or one half inch in diameter and, where they coalesce, a considerable area may be the canker. Old lesions turn brown develop a halo and become corky in appearance, hard and more or less lignified.

The spots may completely girdle smaller twigs and, in that case, the portion beyond the diseased girdle dies and resembles the "die back" of citrus caused by *Colletotrichum.* Leaves badly infected die and the fruit becomes deformed and unmarketable.

Photograph of some leaves of grapefruit with infections caused by *Bacterium citri,* the canker organism.

The organism. It was first named *Pseudomonas citri* by Hesse in 1915, but the next year was renamed *Bacterium citri* by the same author. Then in 1923 it was named *Phytomonas citri* by Hesse and the Society of American Bacteriologists. As pointed out before, however, *Phytomonas* cannot be used for this organism, so that there appears to be a tendency to call it *Bacterium citri* (Hesse) Doidge, dating from 1916 when it was first called *Bacterium citri* by Doidge.

The bacteria are motile by one polar flagellum (see dichotomous key), are 0.5-0.75 × 1.5-20 microns in size, grow in chains and produce capsules. They do not possess spores. They are aerobic, Gram negative, produce neither indol, gas nor acid and do not reduce nitrates. On potato cylinders the growth is somewhat raised, glistening, bright yellow with a narrow white zone around the margin of the colonies which disappears as the surface of the cylinder becomes covered with the bacterial mass.

Citrus Scab

Host plants. The species must severely attacked are; sour oranges, common lemons, rough lemons and tangelos. Some of these that are partially resistant are: king orange, satsuma, tangerine, grape-fruit, sweet lemon and Rungpur lime. The sweet orange is rarely attacked. The citron, some of the mandarin oranges and of the Satsuma oranges are apparently immune.

Geographical distribution. Reported in all sections where citrus fruit is grown.

Appearance on the host plant. The disease appears on the leaves twigs and fruits of the susceptible varieties. For the most part the fungus attacks when the parts are very young and causes irregular, raised, corky lesions which are wartlike, or scablike, from whence it receives its name. On the leaves the disease starts as small, pale, more or less circular spots. These become prominent on one side of the leaf only and on the other side there is a depression. Lesions may appear on both sides of the leaf but usually on the lower side. They may be single or irregularly grouped and may not be visible from the opposite side of the leaf. As these wartlike growths increase in size a corky tissue appears which is at first a pale yellowish orange, later becoming pink and then drab, finally dark olive drab. Fawcett (213) states that the appearance of the scab on the different species of *Citrus* is characteristic. On the satsuma orange the spots are of various shades of red, while on the bitter-sweet orange the border is pinkish buff and in the centre it is umber.

As the wartlike lesions develop a corky tissue appears which is at first pale yellowish orange in colour, later becoming pink, drab and finally dark olive drab. Fawcett (213) states that the appearance of the scab on the different species of *Citrus* is characteristic. The infected spots often run together and thus present a massed appearance. The effect upon the leaves is to cause crinkling and twisting to such an extent that they may not resemble *Citrus* leaves at all.

On the twigs the lesions are found only on the very young tissues but are similar to those on the leaves. If the attack is severe the twigs may be killed.

On the fruit the scabby spots are similar to those on the twigs and leaves but may be more pronounced. If the fruits are attacked when very young they may become misshappen and deformed often falling from the tree. On some of the *Citrus*, especially the grapefruit, the spots may spread over the enlarging fruit until a large portion presents a scabby appearance, thus reducing the sale value of the fruit.

The organism. Elsinoe fawcetti B & J. It was first called *Sporotrichum citri* by Dodge and Butler (Dis. and Pests of Ornamental Plants, James Cattell, Phila, Pum. U. S. A.) Fawcett (213) recorded the fungus as *Cladosporium citri* Massee but Jenkins classified it as *Sphaceloma fawcetti.* However, when the perfect stage was found by Bitancourt and Jenkins (66) it was named *Elsinoe fawcetti* by them. They discovered the perfect stage in Brazil and considered that it was the same as that for the *Citrus* scab organism in the U. S. A.

The conidial stage of the fungus appears on the scabbed areas with the conidiophores arising perpendicularly from the scabby tissue. The conidiophores are cylindrical with sharp pointed apices, one to three celled, hyaline, and measuring 12-20×2.5 microns. The average is 6-8.6×2.5-3.5 microns.

The perfect stage, as described by Bitancourt and Jenkins (66) is as follows:

> *Elsinoe fawcetti* n. sp.
>
> Ascomata more or less scattered, pulvinate, dark brown, circular to elliptical, 38-106× 36-80 microns; epithecium composed of dark coloured pseudoparenchyma, 5.9 microns thick;

> asci 1 to 20 or more in a single ascoma, distributed in the lighter coloured stromatic region beneath the epithecium, globose to ovoid, 12-16 microns in diameter, wall of unexpanded ascus thickened in upper portion; ascospores hyaline, oblong elliptical, 10-12 × 5.6 microns; 2-4 celled, usually constricted at the middle septum, upper half of spore thicker and shorter, lower half thinner and longer; epispore 1.2 microns. Conidial stage *Sphaceloma fawcetti* n. sp.

It would thus be under the *Myrangiaceae* of the *Myrangiales*.

Life cycle. Winston (934) believes that the disease is carried over in the diseased twigs and leaves. The finding of the perfect stage (64) would indicate that it may be the ascospores which are responsible for the spread of the disease in the spring. It may be that the hibernating fungus will produce conidia and these also spread the disease in the early stages of the leaf growth. This would be especially true of the susceptible varieties and species in the neighborhood of the commercial plantings.

The young fruits appear to be extremely susceptible to the fungus, becoming more resistant as they become more mature. By the time the fruits have reached a diameter of 3/4 inch they are mostly immune. On the infected spots spores are produced in large number and scattered by wind, rain and insects. The disease may become epidemic under the optimum weather conditions. The conditions which are conducive to an epidemic are: (*a*) a susceptible host plant, (*b*) young, immature tissue, (*c*) sufficient moisture on the surface of the host tissue, (*d*) temperature between 15-23 degrees Centigrade.

Conidia may be produced as long as the conditions are as above. The ascus stage is a later stage and probably does not contribute to the spread of the disease during the growing season after the first infection.

Control. As the condition for the spread of the disease is of short duration, it follows that any spray schedule must be intensive during that period. Rhoads and De Busk (Florida Ggric. Exper. Stot. Bull. 229 1931) have given the following treatment which proved effective in Florida.

> "If scab is inclined to be severe it is advisable to make two applications of Bordeoux oil emulsion. The first of these (3-3-50 plus 1% oil) should be made just before the growth starts in the spring. The second (3-3-50 plus ½% oil) should be made when about two thirds of the blossoms have fallen. This serves to protect the young fruit."

They found the first application frequently sufficient.

It is not the intention to suggest this formula as one that would give complete satisfaction in India but as a suggestion for experimental work that should be carried on under the local prevailing conditions.

Winston (934) found that of 2 sub-families, 2 tribes, 4 sub-tribes, 22 genera, 35 species, 71 hybrids and 47 varieties studied, only four genera: *Citropsis*, *Citrus*, and *Fortunella* were attacked. Of the genus *Citrus*, however, *C. ichangensis*, *C. medica*, *C. Janos* and *C. webberi* were immune. Peltier (604) found that *C. sinensis* is immune and *C. grandis* only slightly susceptible. He found that the *C. nobilis* group exhibited varied degrees of susceptibility but the tangerine was immune. Scab does not thrive in high temperatures and where the temperature goes above 75 F. for a large portion of the year it is not a problem. There must be at least 50 inches to produce the optimum environment for the fungus.

Gummosis of Citrus

Hosts. The host range is wide, the disease in some form being found on most of the species of citrus as well as on other plants. Among the citrus species the common lime, *Citrus aurantifolia,* appears very susceptible. Reports from California are that the fungi which cause the gummosis on citrus have also been isolated from watermelons, squashes and pumpkins. Mitra (478) reports finding one of the gummosis producing fungi on cotton seedlings and on guava fruit. Ito (259) believes that the fungus which causes the brown spots on tobacco is a biologic form of *Phytophthora parasitica,* one of the important gummosis producing fungi. Tucker (823) has secured isolations of *Phytophthora* from *Antirrhinum, Dianthus, Caryophyllus* and papaw as well as from *Robina pseudoacasia.* Mehrlich reports finding species of gummosis producing *Phytophthora* on potato and tomato. In the case of tomato it caused a serious loss of fruit in transit. Bouriquet found *P. parasitica* on vanilla in Madagascar. Thompson (807) found the same fungus on reoselle in Malaya. *Phytophthora* species which are capable of producing gummosis on *Citrus* have also been reported on castor bean, tobacco, tomato and pineapple. Although this list is incomplete, it gives an idea of the host range of the species of *Phytophthora* which have been found associated with gummosis.

Geographical distribution. Since the gummosis disease of citrus is caused by at least five recognized species of *Phytophthora* it would be expected that it would be widely distributed over the world. Fawcett (213) gives a list of the countries (see latest edition p. 163) in which species of *Phytophthora* have been found causing gummosis. As will be seen, no country which grows citrus has escaped at least one of the various species associated with the disease.

Appearance on the host plant. The disease attacks the trunk and the larger roots. It may extend up into the branches or may be only beneath the surface of the soil. In the susceptible varieties, large patches of the bark may be killed and a considerable amount of gum may exude from the cracks that appear in the diseased area. This is true of the sweet orange, and some of the varieties of lemons and tangerines. In the case of the more resistant varieties the diseased area is smaller and the progress is slower, with a tendency for the healthy area to become delimited from the diseased area. Sometimes there may be distinct ridges at the edge of the canker and in other cases there may be a cracking of the bark at the line between the cankered and healthy tissue with a sloughing away of the dead tissues.

One of the marks of identification of the disease is the gum which exudes from the cracks in the diseased wood. This, however, is not always a diagnostic character as the quantity of gum, or even its absence, may be due to conditions prevailing at the time. Under some conditions the gum may be dissolved in water and washed away. The rapidity of the spread of the disease varies with the variety of the tree and conditions of growth. It often happens that in the early stage of the disease the only external symptoms are the drops of gum which exude, but a close examination will disclose the bark to possess a slightly lighter shade of green than normal.

At Allahabad the disease has been attacking the grape fruit trees which were imported from Florida. Some of these have been almost completely girdled, while others are being attacked in smaller areas only. On the grapefruit trees there is not a large amount of gum exuded although the diseased areas are readily told by the darker color and cracks which appear in the tissues. Sometimes there have been gum pockets formed beneath the tissues of the bark which appear only when the diseased wood is cut away. The specific organism in the graperfruit trees has not been identified as yet so that the details of the disease here may not completely agree with descriptions of gummosis in other parts of the country.

The organisms. At the present time three species of *Phytophthora* are more or less agreed upon as definitely able to cause the gummosis of citrus in nature. They as follows:

Phytophthora citropthora (Sm & Sm) Leonian (374).

Phytophthora parasitica Dastur.
Phytophthora palmivora Butler.

At the same time several other species have been isolated from gummosis areas on citrus trees although they have not been shown to be specific agents of disease in nature:

Phytophthora hibernalis Carne.
Phytophthora syringae Klebahn.
Phytophthora cactorum Sm & Sm.

Recently a number of workers have attempted to classify the *Phytophthora* and, from the studies made, have combined a number of species which, if correct, would include a number of species under the names given above which are found in literature as distinct. Lester Smith (376) considers that *P. faberi* Maubl. should be combined with *P. palmivora* Butler, because of the priority of the latter. Ashby (34) states that there is no real basis for the existence of *P. terrestris* Sherbakoff, *P. melongenae*, *P. allii* and *P. nicotianae* and therefore includes them all under *P. parasitica* Dastur. But he does conclude that *P. parasitica P. palmivora* and *P. colocasia* are distinct and should be kept so. Tucker (823) believes that *P. parasitica* should include *P. melongenae*, *P. allii*, *P. terrestris*, *P. terrestris* var.

rhei, P. jatrophae and *Blepharospora terrestris.* Ito (301) believes that *P. tabaci* Sowada is a biologic form of *P. parasitica.* Leonian (375), whose work has been severely criticized by others, including Tucker (823), merges *P. aracae, P. jatrophae, P. meadii, P. melongenae, P. parasitica,* and *P. tabaci* into *P. palmivora.* Fawcett and Klotz (Calif. Citrograph XXII pp. 64-65 1936) consider that at least four species of *Phytophthora,* i.e., *P. citrophthora, P. parasitica, P. hibernalis* and *P. syringae* may cause the citrus brown rot disease in California. In India the fungi which have been reported include: *P. parasitica,* Dastur, McRae (443), *P. palmivora* Butler, Uppel and Kamat (851) and *P. citrophthora* (Sm & Sm) Leonian by Sharangapani (682). This is not a complete list of the work done on the species of *Phytophthora* found associated with citrus gummosis, but it is enough to give an idea of the present status of the knowledge and classification of the species.

A comparison of the measurements (in microns) of the conidia, oogonia and oospores of three species of gummosis producing *Phytophthora.*

Species	Conidia		Oogonia		Oospores
	length	breadth	length	breadth	
P. citrophora ...	50	35			
P. Parasitica ..	25–50	20–40	18	25	15–20
P. palmivora ..	50	30	20	25	22–24

The mycelium of the three fungi closely resemble each other. In all cases being coenocytic and much branched. In artificial culture the medium has an influence upon the growth of the mycelium. The typical media upon which the fungi make equally good growth are bean meal, Quaker oats and maize meal agar. The diameter of the hypha varies from 4 to 8 microns and the optimum temperature appears to be between 25 and 30°C. In the case of *P. palmivora* it appears to grow best at a temperature of 27-28°. Uppel and Kamat (851) found that the strain which they studied grew at 32.5°C. In the case of *Phytophthora parasitica* the temperature is much higher. It grows well at 35°, whereas *P. citrophthora* has an optimum between 25 and 27°C.

The life cycle. A life cycle for the gummosis organism in relation to the life cycle of the host plants is difficult to relate. However, the life cycle of the fungi may be stated by saying that in most cases the fungus is in the dormant state in the cankers during the dormant season for the trees. When the trees start growth the fungal hyphae begin to penetrate further into the wood in the region of the cambium. Dufrenoy (Rev. de Bot. Appliguee VI pp. 747-754, 1926) found that the fungus of gummosis follows the cambium layer between cortex and wood. Cork layers form which are filled with tannin and shut off the fungus and may even stop the spread of the disease. The conidia are borne terminally on branched conidiophores. They germinate indirectly by means of zoospores, the number varying with the species. In the case of *P. parasitica*, the number is from 5-45, with an average of some 30. The number for *P. citrophthora* averages about 30 and that for *P. palmivora* from 10 to 40. These spores swim about in the water on the surface of the plant and after a time lose their cilia and settle down, forming a distinct wall about themselves to germinate by means of a germ tube which is capable of infecting the host plant. The oospores are not common in nature in trees infected with gummosis and it would seem, therefore, that the fungus lives over in the old cankers.

The infections take place at or near the surface of the ground. It may be that lenticels are a means by which the fungus gains entrance and no doubt injuries caused by insects or other means are also used as entrance ways.

Control. The methods of control recommended are the result of experience and experiment. Common lime stocks appear to be among the most susceptible, although sweet orange is also very susceptible. In some places they are taking steps to prevent the sale of trees grown on sweet lime stocks. Fawcett (213) reports the results of the inoculation or 78 varieties of citrus and found that all lemons are susceptible. Sour orange and two varieties of citron, (*Citrus medica*) were found to be resistant. In Chile, South America, the propagation for commerce of sweet orange (*Citrus sinesnis*) and lemon (*Citrus lemon*) by means of seeds, cuttings, layering or shoots is prohibited. Only trees budded on the sour or Seville orange may be sold.

Soil should be well drained and the trees planted so that the main lateral roots will not be more than two inches below the surface of the soil. Earth should not be piled up around the base of the tree. By careful planting much of the infection can be prevented.

Fawcett (213) recommends that the tree trunks be sprayed with Bordeaux 6-6-50 for the first three feet of trunk and the base of the main branches. He also recommends 12 pounds of zinc sulphate, 1 pound of copper sulphate and 6 pounds of hydrated lime to 100 gallons of water. He also suggests that the dry Bordeaux powder is effective.

In some cases a cylinder of paper is placed around the tree so that it rests on the first main roots. Into this collar a mixture of one part of Bordeaux powder and ten parts of sand, or equal parts of copper sulphate, zinc sulphate and sand are poured. The paper will break and need replacing every year for the first few years until the trees become thoroughly established.

If cankers do occur they may be carefully cut out and the surface of the cut treated with a wash consisting of 1 pound of zinc sulphate, 1/5 pound of copper sulphate, 1 pound of lime and 5 gallons of water.

The fungus will attack the fruit as well, so that a brown rot on the fruit should be viewed with suspicion as it may also be a sign of one or more of the species of *Phytophthora* which cause gummosis.

Corticium Disease of Oranges

Hosts. In India it has been recorded on orange by Dastur (162) and on orange, tea, coffee, *Hevea* and *Cinchona* by Butler and Bisby (96). Stevens (741) reports it on fig, apple, cacao, grapefruit and remarks that some 141 host plants have been recorded for the fungus in the Orient.

Appearance on the Host Plant. The branches and leaves wilt and die with the leaves yellowing and falling. The affected branches are covered with a fine silvery white film of mycelium. Pustules of two kinds may develop in the mycelium, one being white and the other pink. The bark is injured and may either scale off or shred, in which case the wood is exposed. Lightly infected trees may show it only in the cankers that appear on some of the twigs and small branches. Another symptom which is sometimes evident is the cracking and gumming of the branches.

The organism. Dastur (162) has described the fungus as possessing a thin-walled sparsely septate mycelium that grows parallel and interlacing with each other. Entrance to the host plant is generally thought to be by wounds and cracks. The pustules are cushion like, white, pink, orange red or rose coloured. The white cushions are superficial whereas the coloured ones are submerged. In 1941 Dastur (162) described the fungus as *Corticium salmonicolor.* Recently he (165) has changed the name to *Pillicularia salmonicolor* new comb.

The fungus is distinctly a wet season fungus and forms the web over the branches only under humid conditions. Dastur refers to one form as the "Necator" from which produces spores that could spread the fungus during the wet weather. Stromatic masses could also break up into sporelike masses that would be resistant to drouth.

Control. Preventive spraying is done with Bordeaux spray or paste. Dastur also recommends the pruning out and burning the diseased parts.

Sooty Mold

The most common character used to describe this group of fungi is the one used in connection with *Meliola* "sooty mold". The fungi are found on numerous plants in India. Butler and Bisby (96) list seven species which have been reported on the fig, coffee, pine, mango, and other plants. Mundkur (517) records two species, *Capnodium citri* on the leaves and fruits of *Citrus* app. and *Capnodium theae* on the leaves of *Thea sinesis.* It also occurs on sugar cane.

On sugar cane it is not uncommon to find the leaves, or even the whole plant, turned black from the growth of the fungus. Investigation will show that these plants are invariably infested with species of aphis, or other plant insects, which are capable of producing the secretion commonly known as "honey dew" and upon which the fungus grows and fruits. It will grow profusely as long as the honey dew is supplied but quickly disappears as soon as the insects leave. No real damage to the host tissue has been observed but there is no doubt some interference with the photosynthesis and the canes are certainly not attractive for chewing.

The hyphae are brown, septate and broken into two celled segments which are usually 6×12 microns. Pycnidia may be found which are slender elongated structures that may be deeply coloured and open at the top by a fringed ostiole. The pycnospores are small being 4½-6×2-3 microns. Neither Butler and Bisby (96) nor Mundkur (517) list a *Capnodium* sp. on sugar cane indicating that the one on cane has not been named. But, as stated previously, Mundkur lists species on *Citrus* and on tea. As these are all considered sooty molds they will not be discussed separately.

SOME OF THE MORE COMMON DISEASES FOUND ON THE PAPAYA OF NORTHERN INDIA

The papaya, with its soft succulent stems, roots and fruits offers an unusually good opportunity for fungi to attack and the papaya is the victim of some of the worst diseases to be found in the orchard. Among the most common is anthracnose, which may be found on leaves or fruit but is worse on the fruit. Stem and root rot, caused by *Pythium aphanidermatum*, is also important in some cases, even more serious than anthracnose, as the *Pythium* destroys the entire tree. The general root rot, is caused by other fungi than *Pythium aphanidermatum*. Among the fungi isolated from papaya roots have been *Macrophomina phaseoli*, *Rhizoctonia solani*, an unidentified *Pythium* species and species of *Fusarium*. A *Phyllosticta* leaf spot is also common on the leaves of papaya.

Anthracnose of Papaya

Hosts. When all of the various synonyms are taken into consideration the host range becomes an exceedingly wide one, for the various organisms that are associated with the anthracnose disease include those of Citrus die back, mango anthracnose, melon anthracnose, leaf spots of jack fruit, laffa gourd and a number of other plants in addition to papaya.

Geographical distribution. It is world wide. Bryce (89) reported *Colletorichum caricae* on papaya in Ceylon in 1921. Since then the organisms causing anthracnose have been recorded from a large number of countries.

Appearance on the host plant. The first symptoms that are distinctly anthracnose-like are more often seen on the fruit. They appear as small, more or less circular, water soaked spots which vary in size from 1 to 30 mm. As the spots age they become sunken and within the sunken area may be formed concentric rings of acervuli which bear masses of conidia. The spores are colourless when seen individually but often assume a pinkish colour when viewed in mass. They may even become orange coloured. The spots may coalesce and thus form larger areas so that the entire fruit may become one mass of sunken areas each bearing the rings of acervuli.

The rate of spread of the fungus and the prominance of the spore masses will depend upon the weather as the production of spores is correlated with the per centage of humidity. On the petioles and stems the spots assume an elongated shape which extends in the direction of the long axis of the part infected. As the plant ages these spots become covered with acervuli but they are likely to become dark coloured early and rarely exhibit the pinkish masses of spores that are observed on the fruit. On the petioles the acervuli are more likely to be marked by the presence of black setae where as on the fruit the setae are not so common, the difference depending upon the species of *Colletorichum* involved.

The organisms. *Colletotrichum lagenarium* (Pass)
Ell. & Halts.
Colletotrichum gloeosporioides Penzig.
Colletotrichum caricae S. & H.
Colletorichum papayae P. Hen.

The last two are considered as synonyms of *C. gloeosporioides* so that only the first two names will be considered.

Colletotrichum lagenarium (Pass.) Ell. & Holst.

Stevens (741) assigned the fungus to the *Ascomycete* genus *Glomerella* and named it *Glomerella lagenarium* as a result of the production of the ascigerous stage through the use of ultra violet rays. The common anthracnose stage is still called, however, *Colletotrichum lagenarium.*

The spots are usually on leaves but on papayas they are found on leaves, stems and fruits. They are large, roundish, somewhat yellow and may be conspicuously and concentrically zoned. The acervili are small and scattered but usually are on the concentric rings. Spores are nearly colourless when viewed single but may be pinkish to orange when seen in mass. They are typically elongated, ends rounded and with one or more oil droplets visible. See diagram. They measure 13-15×4-5 microns.

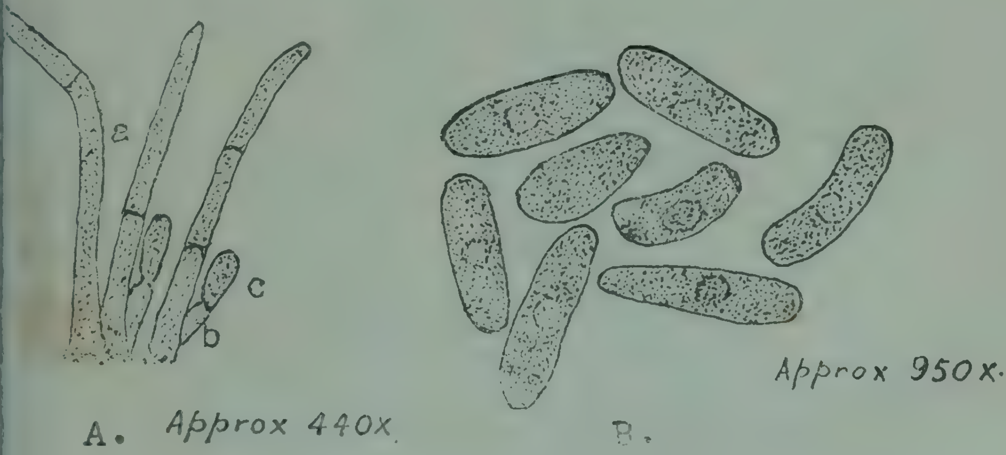


Diagram of *Colletotrichum langenarium* taken from papaya fruit from the Agricultural Institute Farm, Allahabad.

A. a. Setae. b. Conidiophore. c. Conidia.

B. Conidia enlarged.

Setae are rare. When present they are 1-2 septate and from 60-70 microns long. The base is slightly swollen and the tip obtuse.

Colletotrichum gloesporioides Penz.

This species differs from the above chiefly in the presence of large numbers of setae. The sori, which are found more on the petioles and stem than on the fruits, are marked by the black bristly setae. The setae

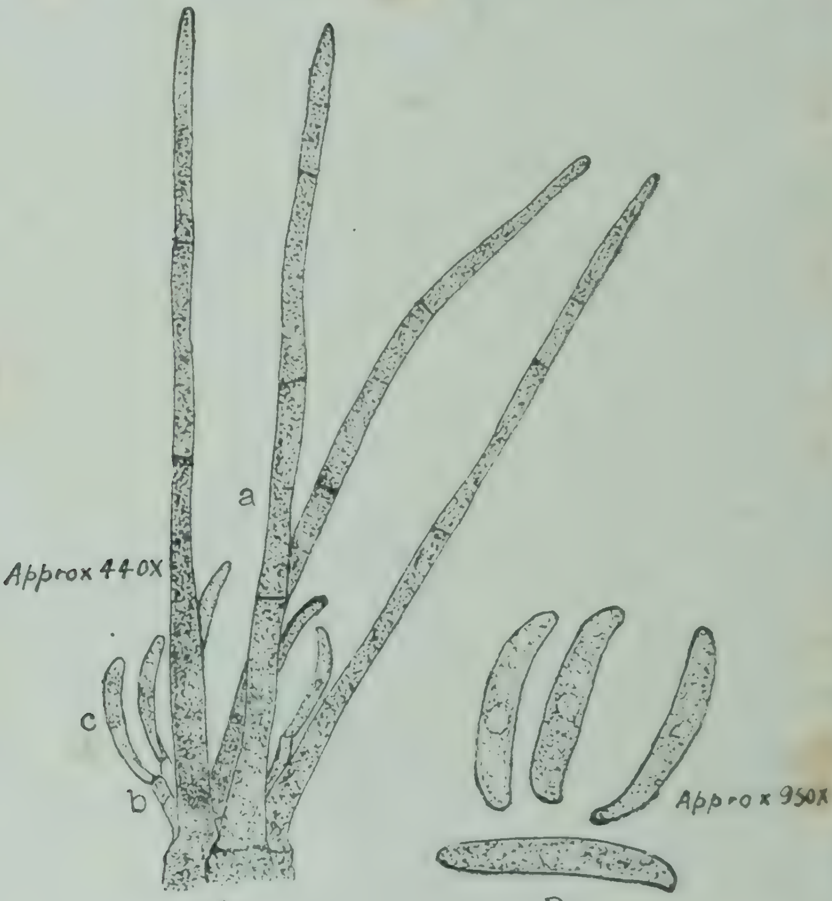


Diagram of *Colletotrichum gloesporioides* taken from papaya fruit from the Agricultural Institute, Orchard, Allahabad.

A. a. Setae. b. Conidiophore. c. Conidia.
B. Conidia enlarged.

are cylindrical, rounded at the apex and line the margin of the acervuli. They are black and measure 40-60 × 5-6 microns. The setae may or may not have septa.

Conidia are straight or may be slightly curved, rounded at the ends and measure 16-18 × 4-6 microns.

Small (713) states that *C. gloesporioides* is related to *Glomerella cingulata.*

Control. So far no satisfactory control has been worked out. Spraying with Bordeaux mixture, or better, with Bergundy mixture together with destruction of the diseased fruits and leaves.

Pythium aphanidermatum (Ed.) Fitz.

Host plants. The host range of *Pythium aphanidermatum* is not nearly as great as that of *P. de Baryanum.* Nevertheless, it is a very serious pest. It has been reported on sugar cane by Bourne on radish by Vaughan, on cultivated *Cucurbitaceae* by Mitra, on *Opuntia, Solanum, Physalis* and *Datura* by Sundararaman, on the roots of cotton by Dastur, on sweet potato and tobacco by Wager, no conifer seedlings by Rathbun Gravatt and the Papaya referred to in this discussion. Recently Yu (946) reported 23 new host plants including tobacco, sweet potato, bean, sugar beet, meloses and cotton. Malik (416) reported the fungus causing collar rot of pigeon pea (*Cajanus indicus*).

Geographical distribution. A glance will show that the reference list represents work done in all quarters of the globe and the fungus has been reported from most of the regions of the world and especially in the tropical and subtropical regions. It was reported in India for the first time in 1935 by Galloway (238).

The organism. The general description of the fungus is very similar to that of *P. de Baryanum* which has just been discussed. The name is under some debate. Most of the mycologists place the fungus under the genus *Pythium.* However, *Sidaris* (691), after studying the taxonomy of the genera *Pythium* and *Nematosporangium*, came to the conclusion that *Pythium aphanidermatum* (Eds.) Fitz. and *P. arrhenomanes* should be renamed *Nematosporangium.* This position was challenged by Sparrow (724) who contended that they should remain in *Pythium.* Sidaris (691) reiterated his stand and stated that the contentions of Sparrow, which are based on the separation of the lobate forms

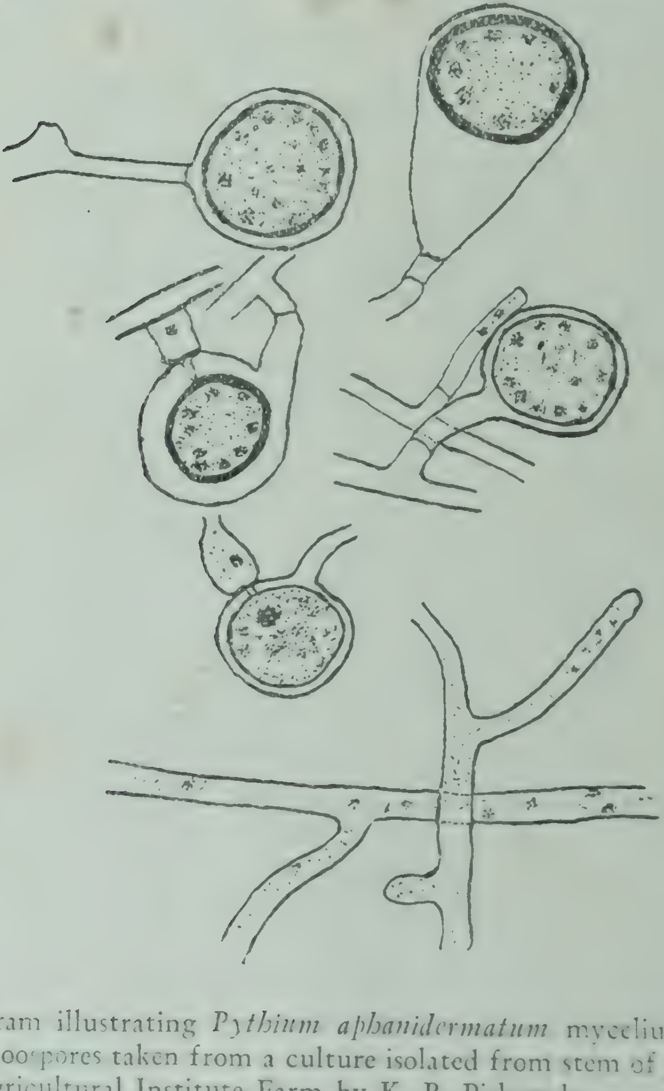


Diagram illustrating *Pythium aphanidermatum* mycelium, oogonia and oospores taken from a culture isolated from stem of papaya on the Agricultural Institute Farm by K. B. Pisharode.

from the filamentous forms, was untenable. Sparrow contended that *Pythium* is the older name and therefore should be given priority. Most mycologists and plant pathologists use the name *Pythium aphanidermatum* (Eds.) Fitz.

In general the sporangia of *P. aphanidermatum* appear to be larger than those of *P. de Baryanum.* Stevens (741) gives the size of the sporangia as 50-100 by 4-20 microns. They are lobate and may have from two to four or more branches when grown on bean agar. Measurements of a number of isolations from papaya at Allahabad are fully as large as those given by Stevens.

Oogonial measurements as given by Drechsler (198) average some 22 microns and compare favourably with the measurements given by Stevens (741). The oogonia are borne on short branches of the mycelium and are nearly spherical with thin walls and dense cytoplasm. Strains from papaya isolated at Allahabad produce a large number of oogonia when grown on the ordinary media. These run from 20 to 27 microns in diameter. Oospores are somewhat smaller in size. They show a very dense cytoplasm and thick walls.

Life cycle. The life cycle of *P. aphanidermatum* is not as well known as that of *P. de Baryannum* but is apparently very nearly the same. In the case of papaya, infection takes place on the roots or stem. If on the stem it is rare that it occurs more than four feet from the soil line and most of the infections on the trees at Allahabad occur within the first three feet of exposed trunk. This also appears to occur mostly on the south west exposure of the trunk, although it may spread around the stem so that the tree may be girdled. It has also been found at Allahabad that insect larvae may enter the diseased tissues and, although this has not been proven, it is quite reasonable to assume that they are a factor in the spread of the fungus within the tree trunks.

The infections may heal over and after the dead tissues have sloughed off leave an open scar. On the other hand, where insect larvae invade the diseased area there is much less likelihood of the wound healing. There is no doubt that in the case of papaya the fungus lives over from season to season in the diseased trees and then as conditions become favourable for conidial formation the conidia are produced as the swarm spores are produced they may be spread by insects and perhaps by birds in some cases. Rain water may also be a factor where the trees are planted close together.

Oospores which are produced in the tissues of the trees may remain in the soil for some time without germination and if the area is replanted reappear in the new planting. This has happened at Allahabad. It is also quite probable that the fungus may live as a parasite on other field crops, although not in evidence, or it may be capable of living a saprophytic life as *P.*

Photograph of a typical papaya stem after attack by the stem rotting fungi of which *Pythium aphanidermatum* is one of the most common.

de Baryanum. The life cycle may thus be worked out as for *P. de Baryanum.*

Photograph of a papaya orchard at the Agricultural Institute, Allahabad, showing the effect of *Pythium* stem rot and other fungi on the stand of two-year old trees.

Control. Little has been done along the matter of control for this specific disease. In general the recommendations for *P. de Baryanum* will also apply but the fact that this fungus can attack trees like papaya above the ground, presents another problem. One of the problems being worked on at Allahabad Agricultural Institute is the control of the stem rotting fungi of the papaya.

There is no doubt that rotation of crops is a very important measure to be considered. Yu (946) found that 1½ to 2% acetic acid at the rate of 2 quarts per square foot of seed bed some 9-13 days before planting the seed controlled the fungi on cabbage, radish and tobacco seedlings. Anderson et al (27) made the generalization that to be effective as a seed disinfectant the fungicide should fulfill the following requirements;

1. Capacity to cover the seed uniformly.
2. Fine enough to pass through a 325 mesh screen.

They further observe that one should not use cuprous oxide in soils with a pH of more than 5 and that seeds of crucifers were injured by the fungicide.

Root Rot of Papaya

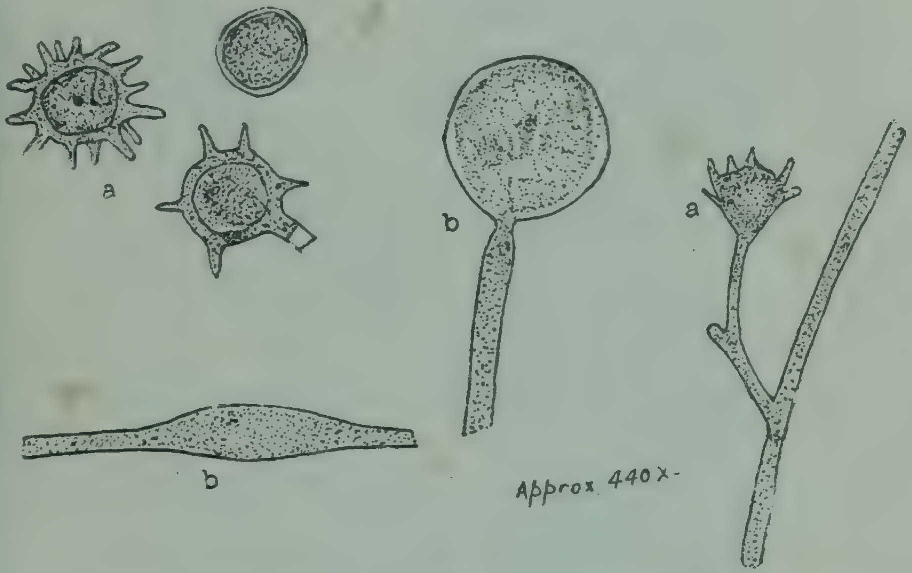
Hosts. The host range is wide for there are at least three organisms that are associated with the root rot of papaya. Each of which is capable of attacking a number of hosts.

Geographical distribution. Wide spread. Probably world-wide.

Appearance on the host plant. The first appearance of root rot may not be readily distinguishable. The rotting may take place during the rainy season and many of the roots may be diseased but because there is ample moisture at that time the wilting and yellowing, which are the main symptoms, may not appear until the dry season.

The first symptoms will be an excessive yellowing of the lower leaves and a reduction in the size of the top leaves. Later the whole top will assume a lighter shade of green and some of the lower leaves may fall prematurely. Fruits will be smaller and there may be a poor set. If trees of this type are dug about at the root and the roots examined they will be found to be attacked by various fungi. Many of them will be found to be decayed and gone. Others will be found to be spotted with disease and where decay has progressed through the root it is soft and mushy. At times nematodes (eel worms) will be found causing root galls and if severe they also cause a loss of colour and low fruit yield. Badly infected trees suffering from root rot appear as the one in the photograph.

The organism. The organisms are, as indicated above, at least three. A *Pythium* (perhaps two) are associated with the root rot. One, illustrated in the following drawing, found on the roots of papayas on the Institute Farm at Allahabad has not been positively indentified. Another isolation was identified by Bhargava (60) as *P. aphanidermatum.*



Diagrams illustrating *Pythium* species found in the roots of diseased papaya seedlings in the nursery at the Agricultural Institute, Allahabad. a. Types of oogonia. b. Types of sporangia. This type appears similar to *Pythium mamillatum* Meurs.

Rhizoctonia solani and at least two species of *Fusarium* have been observed on the decaying roots. In this connection for the former it is not the species of fungi that matter as much as the control measures that are needed.

Control. At this time control measures are not well known. Chemical sterilization might suppress the fungi but would be very expensive. The use of orangic fertilizers may offer some hope. See under Environmental Factors and Plant Disease. Rotation offers little hope as the host range of the fungi is such as to include most of the major crop plants grown on the average farm.

Photograph showing a papaya tree dying from root rot caused by Species of *Rhizoctonia*, *Pythium* and *Fusarium* which were regularly isolated from the roots of such trees. Photograph taken on Agricultural Institute Farm, Allahabad.

Phyllosticta Leaf Spot of Papaya

Hosts. So far it has been reported on papaya and fig.

Geographical distribution. Petch (605) recorded it in Ceylon in 1925 as causing a disease of papaya and it has been recorded from other parts of the world. Infections have been more or less common on papaya at Allahabad during the past two seasons, although no real damage has been done.

Appearance on the host plant. So far as the observations made at Allahabad are concerned it appears the spots are confined to the leaf bases and margins. None were found on the petioles. The spots are rounded to irregular in shape and appear near the edge of the leaf in each case. They are grayish white in the center with a dark margin which may vary from a reddish brown to almost black. In the central portion, which may become fragile and break, may be seen minute black specks which are pycnidia. These are the best means of identification as spots from other causes may resemble those of *Phyllosticta.*

The organism. The pycnidia are dark to black with a distinct ostiole. They erupt through the epidermis and when mature are exposed on the surface. Pycnospores are hyaline, straight, rounded at both ends and range from 2.4-3.6×11.25 microns. They are expelled in a gelatinous mass, cirrus, typical of the members of this genus. Conidiophores are little more

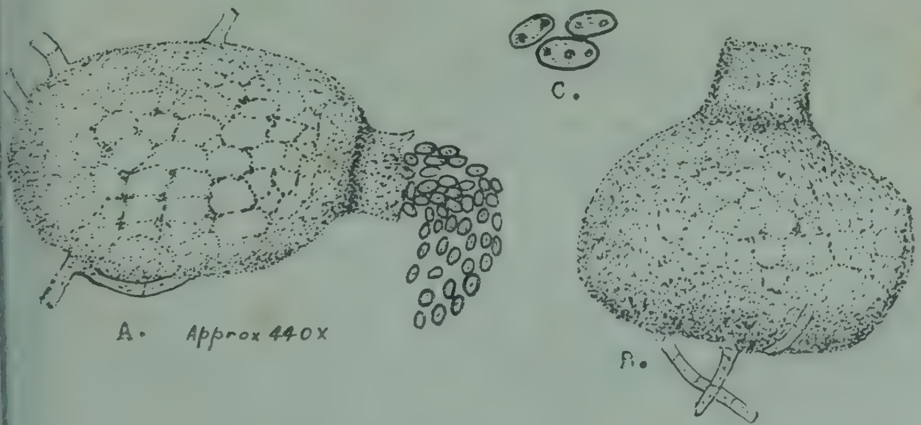


Diagram illustrating pycnidia from *Phyllosticta* leaf spot on papaya, drawn from material collected on Agricultural Institute Farm, Allahabad.

A and B. Pycnidia.
C. Pycnospores.

than basal cells which arise from the bottom of the pycnidium.

Chowdhury reported a Phyllosticta on papaya in Assam but the size of spores which he gave are much too large for the one found at Allahabad. The fungus here is evidently *Phyllosticta caricae papayae* Allesch.

Control. So far the fungus is of minor importance and no serious attention has been given to control.

SOME OF THE MORE COMMON DISEASES OF THE MANGO IN NORTHERN INDIA

The mango is not subject to many diseases. There is a bacterial disease that occasionally does some damage. Mango anthracnose occurs some times. Since it is caused by the same organism that causes wither tip of *Citrus* sources of infection are usually near at hand. Sometimes powdery mildew has been reported on the mango and a seedling disease caused by *Gloesporium mangiferae* is sometimes present. A black tip, which has been thought to be due to smoke from brick kilns, and other such coal burning industries, has been reported on mangoes.

Bacterial Disease of Mango

Host. It appears to be confined to the Mango.

Geographical distribution. South and East Afric and Egypt. More recently it has become serious in Palestine.

Appearance on the host plant. It occurs mostl on the fruit, although it also appears on stems, twig and leaves. On the leaf the lesions are angular, wate soaked spots. While they do not become large independently, yet they may coalesce and form areas tha soon become brown, are slightly raised, shiny and ofte with an exudate of gum. In addition, on the old leaves the spots become dry, gray in colour and late crack and fall out. On the stems, the lesions are th cause of longitudinal cracks. They occur on all parts of the stem but most commonly on the young rapidly growing tissue. They are usually associated with gummosis. Infection in the inflorescence causes the pedicels to become blackened and the fruit to fall. On the fruit the spots are initiated by wounds and become blackened and the fruit fall. On the fruit the centres of spots are usually white and water soaked. They spread irregularly and may become as much as 15 mm. in diameter. Gum exudes from the lesions on the fruit and runs over the fruit to the next below, and thus in rainy weather is a source of infection to all below.

The organism. It is a motile rod with two to eight peritrichous flagella. As it produces no spores, it cannot be called a *Bacillus,* as was done by the Society of Americans Bacteriologists. It is a Gram positive, aerobic or facultative anaerobe. It produces no gas and only slight acid from lactose, sucrose, levulose, dextrose. It produces ammonia and indol and on potato cylinders it produces a yellowish colony. The optimum temperature is 80°C. and the thermal death point is 60°C.

Following the report of the Nomenclature Committee of the Second International Society for *Microbiology* it would appear that the name of the organism should be *Erwinia mangifera* (Doidge) Bergey et al.

Control. Wager (904) reported that in South Africa it was found that Bordeaux mixture used at standard strength, with a good spreader, was sufficient to produce clean fruit. When seasons are wet, the damage is severe and the necessity for the spray is increased. Gathering and burning all of the diseased fruit is also recommended. In Palestine it has been legislated against (Govt. of Palestine Plant Prot. Ord. 1924) as one of the serious diseases.

Mango Anthracnose

Hosts. Glomerella cingulata (S. & VS.) is found on a wide range of host plants which include *Capsicum* spp., coffee, various species of *Citrus* and *Malus* as well as other plants of economic importance.

Geographical distribution. It is world-wide wherever the hosts are grown. In India it is found on chillies, species of *Citrus* and the mango.

Appearance on the host plant. It may appear on the leaves, petioles, twigs and fruits. On the fruits irregular brown spots of variable size form. On the leaves the infections may begin at any point and may spread so that the entire leaf is included. The necrotic areas are elongated and the tissues may rupture. Young leaves are more severely attacked than the older ones. Infected petioles turn gray and the leaves wilt, finally falling, leaving a black leaf scar. On the twigs the fungus produces elongated black necrotic areas. The young twigs are the first to be attacked and dry from the top to bottom. When severe the young twigs may all die and thus the disease resembles the Die Back of *Citrus* which is caused by the same organism. The fruit spots on the fruit become black followed by a rot which may rapidly envelop the whole fruit.

The organism. Colletotrichum gloeosporioides Penzig. Acervuli may be seen in abundance on the diseased portions of the plant. They are brown to black in appearance. On the leaves they appear on both sides. At first they are sub-epidermal but later they rupture through the epidermal tissues and develop masses of pink spores which are straight, cylindrical or slightly oval, measuring some 18×4-6 microns. The setae are few, cylindrical, rounded at the apex and are found mostly on the margin of the acervuli. They contain few septa, are fuliginous (smoky) black and measure 4-90×5-6 microns. The conidiophores are distinct and hyaline. The organism may remain viable on old leaves, in contact with the soil, for some two years.

Control. For the present the best control measures appear to be the collecting and burning of the diseased plant parts. This means cooperative action. Spraying with Bordeaux 3-3-50 is also recommended. This should be done during the months of February, April or September (554).

Powdery Mildew of the Mango

Hosts. *Mangifera indica.*

Appearance on the host plant. General appearance of the powdery mildew and one time was assigned to the species *Erysiphe cichoracearum.*

The organism. *Oidium mangiferae* Berthet.

The mycelium is branched, hyaline, superficial and composed of septate hyphae which are 4.1—8.2 microns (Uppal et al 860) Conidiophores are simple, erect, 64-163 microns with two or more basal cells. Conidia unicellular, hyaline, elliptical, 25-48.9×16-23.9 microns. They are produced singly or rarely in twos.

Control. Uppal (860) reported some damage along the coast during cool weather. Where serious, sulphur dusting would not doubt help.

Gloeosporium mangiferae on Mangoes

Muller (506) has reported *Gloesporium mangiferae* on mangoes in the Dutch East Indies where it attacks the seedlings and causes a damping off. It has also been found on the fruits, twigs and inflorescences of the older trees. He reported control with 1.5 per cent Bordeaux.

Black Tip Disease of the Mango

Hosts. This disease appears to be confined to the mango although it may be on other fruits but under different names.

Geographical distribution. Limited to parts of India.

Appearance on the host plant. Sen (678) made the first record of this disease in 1946 although it may have been observed before and associated with some other trouble. When the fruits are about one inch long the outer tip begins to yellow and later to turn brown and then black. The blackened end dries and the drying may include the whole of the fruit except a little at the stem end. The disease always starts from the tip and progresses upward towards the stem.

The general opinion has been that brick kiln smoke would cause the disease and the injury decreases with distance from the kilns. Some damage has been found as much as 700 feet from the kiln.

When coal smoke was artificially applied to the fruit it produced the tip injury thus verifying the general idea of the cause. It was determined that sulphur dioxide, ethylene and carbon monoxide caused the injury.

Control. A smoke screen in the chimneys would aid in preventing the injury.

Red Rust of Mango

Host plants. It has been reported on the mango, jujube, persimmon, guava, acacia, jasmine, magnolia, rhododendron and a number of other plants.

Geographic distribution. It has been recorded in a number of the tropical and semi-tropical countries.

Appearance on the host plant. The rust, which is not a rust at all, appears as rusty red blotches on the leaves, uaually the upper surface. They are small at first, later they may become as much as a half inch in size. The leaf tissue is uninjured but the rust interferes with photosynthisis and in this causes an indirect injury.

The organism. Cephaleuros virescens is a re algae and not a fungus. It is usually distributed in the zoospore stage during wet weather. Chester (105) states that it has been recorded as serious in the southern portion of the U. S. on some plants.

Control. Where it appears serious Bordeaux mixture will control. It is of so little importance that usually there are no recommendations for control.

SOME COMMON DISEASES OF THE APPLE IN NORTHERN INDIA

Apple growing is confined to the higher altitudes of India and thus in the cooler regions. In several cases the fungus is limited to the species of Pyrus so that is not widely distributed in India. One of the most wide spread (in relation to the world) of the apple diseases known in India is apple canker. It probably was introduced into India from outside. Two diseases that are not widely known are the stem brown disease and the stem black disease. Powdery mildew is common some seasons. Fruit rots are some times serious. Soft rot caused by *Penicillium expansum* and a species of *Rhizopus* may do serious damage in some cases.

Canker of Apple Trees

Mundkur and Keshwalla (527) report that apples received from Australia developed the canker caused by *Sphaeropsis malorum* Pk. This is the only report to date of that fungus in India.

Hosts. The fungus is widespread on various hosts over the world. Especially of those belonging to the pome fruits.

Geographical distribution. World-wide in the more temperate regions.

Appearance on the host plant. On the stems the cankers are elongated in the direction of the long axis of the stem. The bark has a tendency to crack, wrinkle and blister with the outer layers peeling off.

It is believed that the infections are most often via wounds. Spring infections are believed to occur more often than at any other time. On the fruit the disease is often called a brown rot. The infections are likely to be at the blossom end. Pycnidia occur over the diseased surface giving it a roughened appearance.

The organism. *Sphaeropsis malorum* Pk. The mycelium within the host tissue is sooty brown to olivaceous. Pycnidia are erumpent, usually surrounded by the broken epidermis and in cross section they appear somewhat depressed-conical in shape. The spores the oblong-elliptical, brown and usually about twice as long as broad, measuring some 22-32×10-14 microns. The size varies with the host attacked.

Control. Pruning and destroying the cankers at the same time using some fungicidal paint for the wounds.

Stem Brown Disease of Apples

Hosts. Normally reported on currents. Singh (706) records it on apples.

Geographical distribution. The disease, like the stem-black disease, begins with the pruned branches and proceeds dwonward, causing a type of die back. In this disease the upper limbs are more liable to attack than the lower ones. As the disease progresses the bark is loosened, becomes papery and brown and rolls outward. When the bark is removed it is found that the wood is stained dark brown. At the same time there are likely to be found longitudinal fissures. The disease becomes evident about the last week of April and is most severe during May.

The organism. *Botrysphaeria ribis* Gross.

Perithecia are rarely found in nature. Pycnidia are found just outside the cork cambium. There are two types of pycnidia; Type A and Type B.

Type A. These are minute, measuring 0.25-0.5 mm. The centers are more or less solid pseudoparenchyma tissue with cavities here and there in which spores are found. Pycnospores are bicillar, allantoid, measuring 9-12×1 microns. They do not germinate.

Type B. These occur singly or in groups of 2-6, are globose, black, erumpent, ostiolate and possessing a thick wall. They measure 126-394×114.8-210 microns. The average being 220.7×166.3 microns. Fertile hyphae are small, unbranched, hyaline, bearing pycnospores at the tip. The pycnospores are fusoid to oblong, elliptical, hyaline to subhyaline, unicellular and measure 9.1-25.6×5.6-7 microns. These germinate readily.

Perithecia, when found, are round tipped with papillate ostiole. They occur singly or in groups of 2 to 8. They measure 140-252×127.4-280 microns with the average 190.7×192 microns. Singh (Ind. Journ. agric. Sci., XII pp. 368-380 1942) has given the above measurements for the fungus he studied in the Kumaun.

The asci are clavate, 8-spored, hyaline and measure 36.4-112.0 microns×14.0-18.9 microns. The average being 77.5×15.4 microns. The relation between the ascospores and the spread of the disease is not clear.

Control. The control measures are not given but it is clear that sanitation and use of some antiseptic paint at time of pruning is essential.

The Stem-Black Disease of Apples in the Kumaun

Host. Apple in the Kumaun area.

Geographic distribution. In the Kumaun district, India.

Appearance on the host plant. The first symptoms of the disease is a cracking or black streak which spreads downward from the pruned surface. These extend around until they girdle the branch killing it. The streaks result in cankers that extend through the true cambium into the xylem wood beneath. The disease starts in July and reaches its highest infection stage in August.

The organism. Conothecium chomatosporum Corda.

The disease was first reported in the Kumaun region by Shaw in 1918 and by Butler in 1919. The pycnidial stage was first reported by Massee and Van der Bijl in 1915, the *Conothecium* stage being the chief one seen. The pycnidial stage has not been reported from the Kumaun.

The hyphae are short and appear conidia like. Conidia are irregular, circiniform, muticate and often coalescing. They are dark brown in colour; measure 8.4-36.4×5.6-30.8. The average being 16.3×11.4 according to Dey and Singh (187).

Control. Control measures consist of careful pruning, using a good fungicide paint. For the paint, use 2 oz. red lead; 2oz. copper carbonate; 100cc. raw linseed oil. Wind and water are considered to be agents in the spread.

Podosphaera leucotricha (E and E) Salmon.

Hosts. Butler and Bisby (96) report the mildew on apple in Kashmir. It has been reported from every apple growing country of the world. It seems to be confined to the species of the genus *Malus* or *Pyrus*.

Geographic distribution. It has been reported in Tasmania (96), in Australia (96) in United States, Japan and Canada. In some sections the disease has become severe on apple trees.

Appearance on the host plant. It is found on leaves, twigs, flowers and fruits. It first appears as small grayish or white flesh like patches of fungus growth. It appears on the under side of the leaves first, which soon curl and then become folded the long way. The diseased leaves become hard, brittle and often crack. The deformed leaves thus are unable to do the normal work and as a result the younger portion of the growth is stunted and later killed. Apple trees often show a number of bare branches and the top which are the result of the powdery mildew attack.

If the buds are attacked they may be killed or they may remain dormant and the fungus will live over in them. Fruits will be attacked after the petals have fallen and may be killed or reduced in size and the surface may show the effect of the fungus. A russeting of the skin is evidence of such an attack and, if severe, the skin may crack (which) permits secondary fungi to attack and cause early decay.

Thus there are several symptoms which are characteristic of the fungus; (1) on leaves the fungus causes curling and stunting: (2) on twigs the leaves are stunted and killed leaving bare branches: (3) flowers are deformed and fruit fails to set: (4) buds to produce the fruit and foilage for the following season are killed: (5) fruit is russeted, may crack or may not mature.

The organism. The fungus is known as *Podosphaera leucotricha* (E. and E.) Salmon. It is very similar to another species, *P. oxycanthae* (DC) de Bary, and is often confused with it in the field. It is a more serious parasite than *P. oxycanthae* and is the only one of the two mentioned in India at this time.

The fungus, as a parasite is entirely dependent upon its host and thus there is no question of the causal agent. Infection may occur as a result of the activities of either ascospores or conidia. Germination of either type of spore sends a thread of mycelium over the surface of the host tissue and soon haustoria are penetrating the host cells and shortly after, numerous erect branches are produced which bear at the tips chains of barrel shaped spores or conidia. These conidia are capable of immediate germination and further infections can be immediately spread.

By the middle of the growing season the surface mycelium has changed from the colourless condition to brown and typical oogonia and antheridia are formed. Following fertilization, perithecia are formed which are as described under the genus *Podosphaera* above.

The single ascus within the perithecium measures some 55-70×44-50 microns. The ascospores are hyaline and single celled. They are forcibly expelled and may be carried by the air currents to distant points. The perithecia are confined to the twigs and petioles, or more rarely may be found on the midribs and large veins. It is generally held that the fungus remains dormant as mycelium in the buds and not as ascospores. That is, the new infections are not from ascospores but from the dormant mycelium in the buds and tender twig portions.

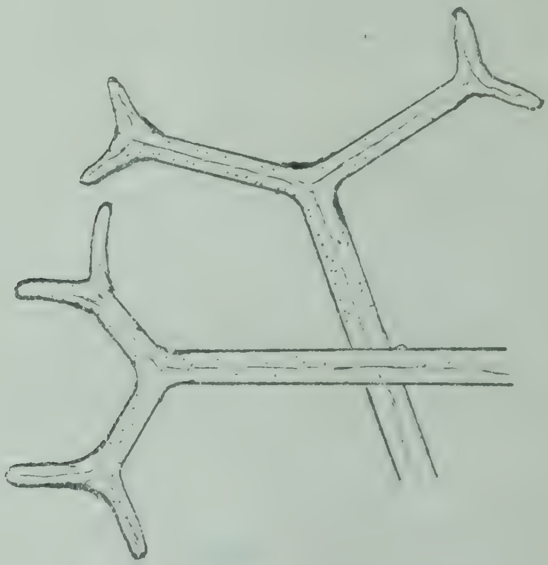


Diagram illustrating the appendage tips of the powdery mildew of apple which is found in India, *Podosphaera leucotricha*. The ascocarp is very nearly identical with that of *P. oxyacanthae* (D. C.) de Bary.

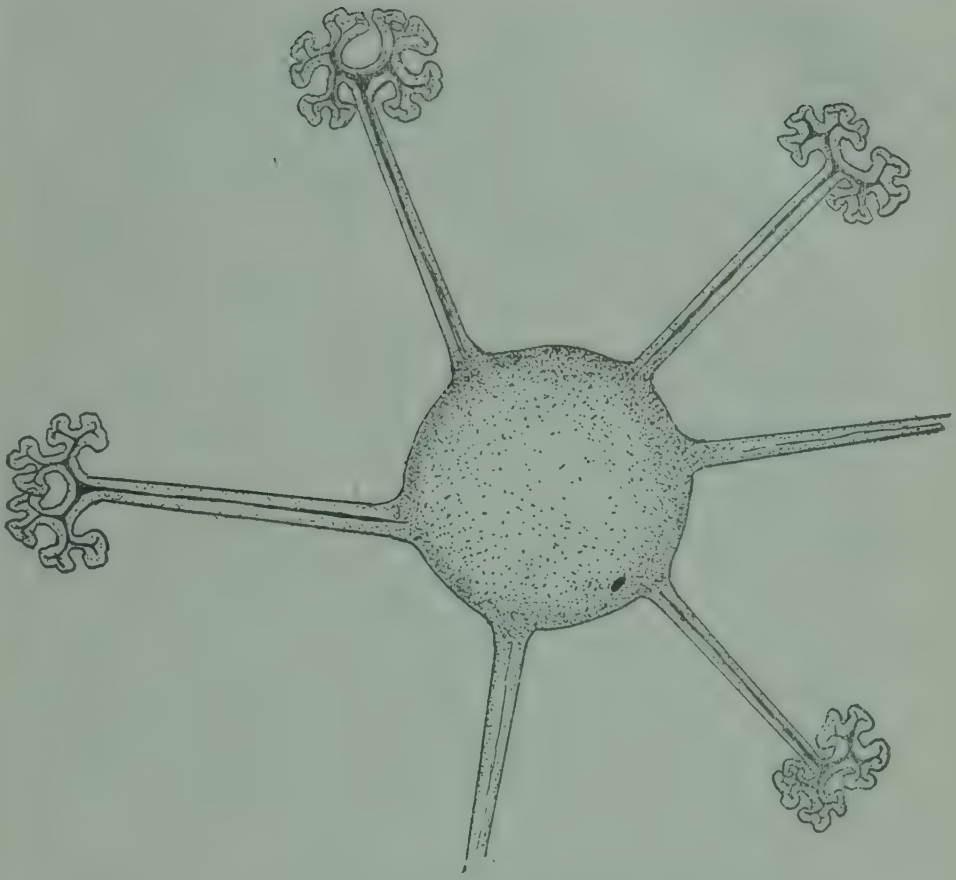


Diagram illustrating the ascocarp of powdery mildew of apple, *Podosphaera oxycanthe.* This species is not found in India at present. Tip of the appendage is different from that of *P. leucotricha* shown above.

Life cycle. The life cycle is simple. Dormant mycelium, or more rarely ascospores, initiate the early infections and soon conidia are formed which spread the disease throughout the growing season. At the close of the growing season the dormant mycelium and the ascospores carry the fungus over the winter season.

Control. Barss (46) recommends sulphur dust for the control of the mildew on apple and pear. This is especially effective in warm weather. A now "micro sulphur" has been recommended for the control of the apple mildew. By adding ground brimstone the control period was extended to some three weeks as compared with the ten days of the free sulphur alone. Thomas (802) reports the control of *P. oxycanthae* by the use of atomic sulphur in Tasmania. Brereton (80) reports the control of the mildew on apples in New South walves by the use of hydrated sulphur. Heald (75) gives a very good discussion of the mildew in the United States. He recommends pruning off the diseased shoots to remove the dormant fungus; study or cultural conditions to keep the trees in the best of condition; and spraying with lime sulphur (1-50) or iron sulphide at the time the buds are just beginning to turn pink, when the calyx is just closing and two or three weeks after. The 4-4-50 neutral Bordeaux, copper carbonate (3-5-50) or Burgundy mixture (4-5-2-50) may also be used but it is suggested that in the use of these various sprays it is best to test the sprays before using and select the one that is best adopted to local conditions. Thomas (802) reports that the addition of iron sulphide improve the colour of the trees.

Soft Rot of Apples

Hosts. Wide range. Most fruits.

Geographic distribution. World wide.

Appearance on the host plant. The diseased areas on the fruit turn soft and become watery, later turning light or yellowish brown in colour. Singh (704) states that in the larger areas the skin becomes wrinkled, often forming somewhat concentric rings of wrinkles. Primarily a ripe fruit rot, the fungus gains entrance mostly through the stem end, although it may gain entrance through wounds at any point.

The organism. Penicillium expansum (Link) Thom.

Conidial fruitfication is typically in three stages.

The primary branches are 130-200×50-60 microns, consisting of one to three primary branches which bear the secondary branches that are crowded together. These are 8-10×3-3.4 microns and bear chains of conidia which are 2-3.3×3-3.4 microns. Gilman (248) reports it from soil in numerous places in the United States and other countries. Singh (704) recommends Bordeaux as a control.

Rhizopus Rot of Apples

Geographic distribution. Mehta (457) reports that a fruit rot of apples is caused by a species of *Rhizopus* which has apparently not been observed in the hill country of India before. Species of *Rhizopus* have been reported to cause spoilage of fruit in other parts of the world but the fruit has been of the soft fleshy type, such as berries, peaches, etc.

Appearance on the host plant. The fungus does not show on the surface until the skin is broken. On the light skinned apples, such as the "Kulu" variety, the skin over the diseased portion may become russet coloured to brown and is easily peeled away. The fruit under the skin is discoloured a zinc-orange colour with a slightly sour taste but a not unpleasant odour. In some cases the skin will wrinkle and an exudate will be given off.

The organism. The fungus rarely shows on the surface but the carpellary cavities are likely to be filled with the mycelium. If the skin is removed the fungus will appear on the surface within 24 hours. The mycelium is composed of broad, aseptate hypha, with few intercalary hyaline chlamydospores. Rhizoids are rare. Sporangiophores are borne on bulbous brown swellings 40-80 microns in diameter. The sporangiophores are one to many, range from 160-480×10-12 microns. The sporanga are dark, more or less globose in shape, measure 80×176 microns in diameter. The columella is more or less oval, measuring 60-100×

66-112 microns. The spores are light gray, plain surfaced, slightly angular and faintly striated, measuring 5-7.2 microns.

The fungus was identified as *Rhizopus arrhizus*, Fisher.

Control. So far no control has been worked out. Light coloured varieties are more susceptible than the dark skinned ones but as some of the best apples are light skinned it is not suggested that they not be grown.

SOME OF THE MORE COMMON DISEASES OF PEACHES IN NORTHERN INDIA

The peach is grown in the higher altitudes along with the apple and the pear. There are a number of diseases common on the peach of which the most common is the peach leaf curl. This is severe in some seasons and complete defoliation occurs. Powdery mildew is common in some sections. However as further reports of this fact have not appeared it may be a mistake. Brown rot has also been found in the peach in Kashmir Fruit. Rots caused by Rhizopus, Penicillium and Aspergillus are also common although little fruit is actually stored so that damage is not severe. In 1936 Keshwalla (332) reported scab, a leaf spot caused by a species of *Coniothecium* and another leaf spot caused by *Phyllosticta prunicola* Padwick (575) reported that *Phytophthora parasitica* had been found causing a rot of peach.

Peach Leaf Curl

Host plants. This disease is common on the peach and nectarine as well as the almond.

Geographic distribution. It is found generally wherever the hosts are cultivated.

Appearance on the host plant. The disease appears on the tender leaves soon after they unfold and causes a wrinkling and deforming from which it receives its name. The disease may infect any portion of the leaves and is usually accompanied by a lighter colour, a darker colour or even a reddening of that portion. Badly infected leaves fall and replacement takes the strength from the tree so that the fruit is smaller and less highly flavoured.

Infection is not confined to the leaves but may occur also on fruits and twigs. Where the fruits are attacked they may become dwarfed and unequally shaped. These fruits usually fall before maturity.

Losses from the disease may be extremely heavy. In the United States it has been estimated that the losses may be as much as $5,000,000 per season from peach leaf curl alone.

The organism. The organism is generally known as *Taphrina deformans* Tul. as it was first described by Tulasnein 1866. Three years later Fuckel described it as *Exoascus deformans.* Since then plant pathologists have been divided in the use of the names. Mix (499) following Sadebeck in his classification of the *Exoascaceae*, referred to the organism as Exoascus *deformans.* Butler and Bisby refer to it is *Taphrina deformans.*

The mycelium of the fungus is intercellular and may be found in the spaces between the cells of the diseased leaves. Several types of hyphae have been recognized such as; vegetative hypha, that which is found in the parenchyma tissues; distributive hypha, that which is found in the pith of diseased plant parts; and the fruiting or reproductive hypha, that which develops into chlamydospores and then into asci. The question of the life of the mycelium is discussed by Mix (499) and he is convinced that there is no such thing as perennial mycelium in *T. deformans*, although he says that other species do possess perennial mycelium. He believes that the fungus lives over winter as conidia which may adhere to any portion of the stem or branches.

Mix (499) believes that most of the infections take place through the lower surface as the upper surface is protected by the folded leaves. The conidial germ tubes may penetrate the thinly outlined walls of the young cells. To what extent the development of cutin on the older cell walls may act as a barrier to the penetration of the germ tubes is not known. Once inside, the hyphae may grow in any direction where they find intercelluar openings.

The conidia are uninucleate when produced. In referring to conidia in this case it is really the ascospores which are meant. Although the copulation of these spores has been suggested, Mix (499) was unable to convince himself that it is a general thing. He found some very definite copulation and the spore receiving the nuclear material put forth a germ tube, but on the other hand there were so many spores that germinated without copulation that he was convinced that copulation did not occur regularly in *T. deformans*.

The formation of the ascus in *T. deformans* is as described above for the *Exoascaseae* and will not be repeated here. The ascospores bud to form conidia and these are the spores which carry over the cold season.

Conidia survive the resting period on various parts of the tree and, with the swelling of the buds in the spring, they become alive. As they chance to be placed on the young tender leaves they germinate and infection follows. Asci soon form and the ascospores are widely distributed in rain and wind to other leaves and tender portions of the plant. Continued infection soon carries the fungus to all parts of the plant, and, if climatic conditions are optimum for the fungus, there may be a severe epidemic which will destroy all of the leaves. As the end of the season approaches the conidia resulting from the budding of the ascospores are distributed and find lodging on various parts of the tree. Thus the life cycle is from conidium to infection in the early part of the growing season. With the introduction of ascospores, continued infections throughout the growing season and the production by budding of conidia which act as the overwintering agents.

Control. Just as the buds begin to swell, the application of 1 : 40 lime sulphur or 4-4-50 Boardeaux wll do much to control the disease. A foll spray with which ever fungicide is better suited to the orchard conditions is also an aid.

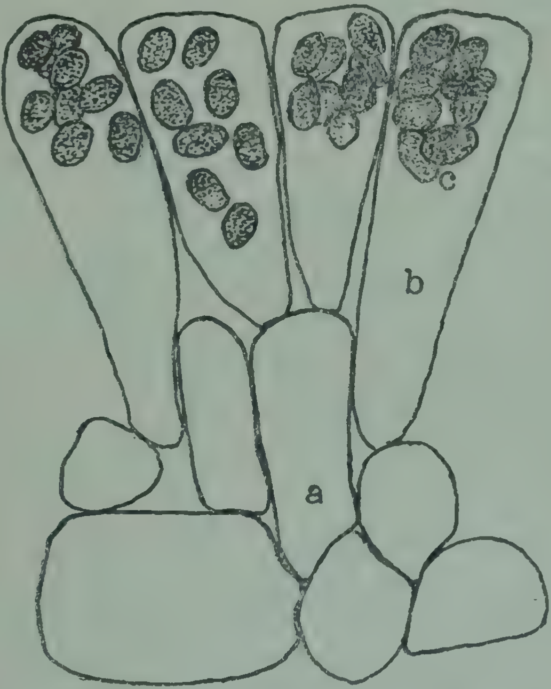


Diagram of asci and ascospores of *Taphrina deformans* which causes peach leaf curl.

a. Host tissue.
b. Ascus.
c. Ascospores.

Powdery Mildew Of Peach.

Hosts. It has been reported on the almond and peach in Kashmir, Darjeeling, Belgaum, Simla and Dehra Dun. It has also been reported in Bombay and Nagpur.

Geographic distribution. In addition to the places in India it is found over much of the temperate part of the world.

Appearance on the host plant. The powdery mildews are named from the powdery appearance they give to the plants they grow on. This powdery appearance is caused by the numerous conidiophores which arise from the surface of the host and the many conidia they produce. The mycelium is superficial as none of the hyphae penetrate beneath the epidermis (epidermal layer). At the end of the active growing season of the host the fungus ceases the asexual reproduction and may then produce the sexual stage. After the union of gametes there are produced round fruiting bodies (ascocarps) which appear as tiny black dots on the surface of the mycelium. These are closed bodies (cleistothecia) containing from one to several typically 8-spored, asci.

The organism. *Sphaerotheca Pannosa* (Wallr.) Lev.

The conidiophores are erect hyphae which arise from the prostrate mycelia on the surface of the host plant. Conidia are pinched off from the tips in succession so that they form a chain of short, barrel shaped cells, the terminal one of which elongates slightly, rounds at the ends and falls. They germinate readily and are the source of further infections. The fungus feeds by means of hasutoria which are forced into the epidermal cells.

Sexual reproduction is by ascospores which are formed as a result of the union of male and female gametes. Following this union there is produced a round mass of mycelium within which is contained one ascus that is typically 8-spored. The appendages are hypha like and flexuous.

Control. Bordeaux mixture, copper oxide or sulphur sprays are recommended.

Brown Rot of Stone Fruits

There is only one report of brown rot being in India and that may be a mistake. However, the disease is so common in other parts of the world that it is included here.

Hosts. All of the stone fruits, such as the peach, apricot, cherry, plum, nectarine etc.

Geographic distribution. Found generally over North America and Europe.

Appearance on the host plant. The fungus can ttack the blossoms and twigs, as well as the fruit. The blossoms are blighted and blackened. Twigs killed re often the fruit spurs and thus the bearing wood s reduced. Fruit rotting is the most common form of ttack. The fruit is infected through wounds, such as nsect bites, abrasions by limb rubbing or the rubbing f two fruits together in a cluster. As the fruits rot hey take on a brown colour in the decaying area. Soon he decaying areas are marked by concentric rings of rayish coloured masses of fungus growth. These masses vill be composed of large numbers of conidia and onidiophores. See diagram. These conidia are scattered by wind or water and if a rotted fruit should ppear first in the tops of the trees the fungus soon preads over all of the tree as the spores will be splashed n the rain. As the fruits, decay some of them fall ut many will remain on the tree and these dry up and orm mummies. Within these mummified fruits is a mass of mycelium or sclerotium. These are a mark of the disease.

The organism. There are several species of the brown rot.

Sclerotinia fructigena (Pers.) Schr. known over Europe.

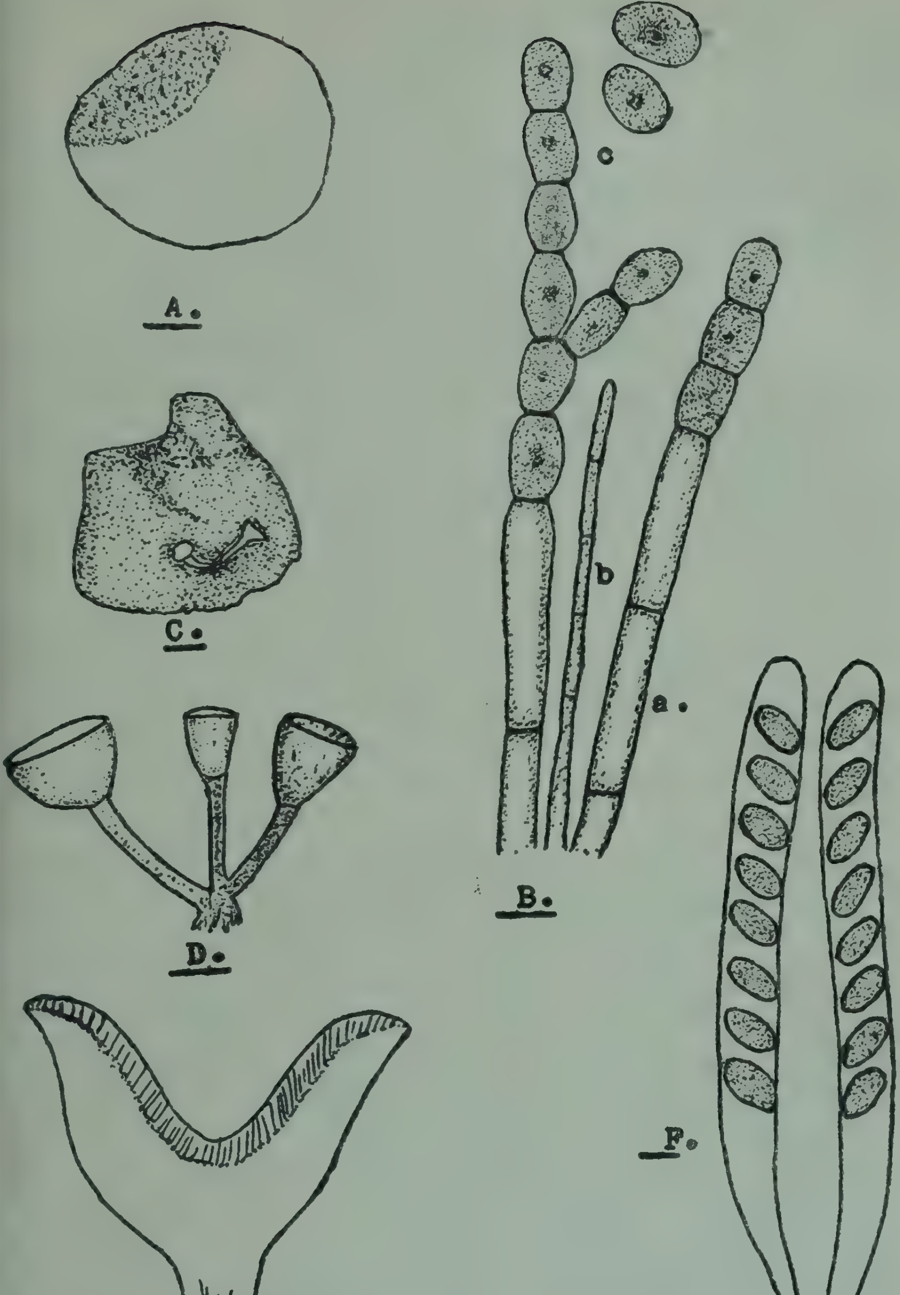
Sclerotinia cinerea (Bon.) Schr.

There are several forms of the species. Forma *mali* is known in Europe. Forma *pruni* is known in England, Europe and America.

Sclerotinia americana (Worm.) N. & E. is known in America, Australia and New Zealand.

The life cycle of one of the species is very similar to that of each other species. As mentioned above the conidial masses are a mark of identification on the diseased fruits. Those of S. *fructigena* are yellow buff in colour with conidia that are some 21-13 microns. The conidial pustules of S. *cinerea* are gray in colour. The conidia are smaller, being 17-11 microns. The conidiophores are short, septate, and cut off conidia from the tips. The conidia are cut off successively so that they form chains with the outermost maturing and falling. These conidia are carried from place to place by wind and rain. Insects may also play a part.

Mummied fruits that fall to the ground are covered by leaves and soil and remain dormant for one or two seasons. If the conditions are right for germination the following season at the time of blossoming, the mummied fruits produce cup shaped fruiting bodies which bear in the inside a fruiting layer (hymenium) on which are borne the asci. These are cylindric bodies which are typically eight spored. As the blossoms of the peach, apricot or plum open the same climatic conditions cause the asci to explode and fire the ascospores into the air. These are carried to the blossoms and infection takes place with blossom blight as a result. Conidia are then produced and the cycle of summer infection is on again.



Diagrams illustrating brown rot (*Sclerotinia fructicola*).
A. Diseased fruit.
B. a. Conidiophore. b. Paraphysis. c. Conidia.
C. Mummied fruit showing apothecia.
D. Apothecia.
E. L. S. of an apothecium.
F. Asci with ascospores.

Control. Sanitation. Destroy the old mummies and prune out the diseased twigs. Fungicides, such as Bordeaux, or wettable sulphur may be used. Chester (105) suggests a dust composed of 80 per cent sulphur, 5 per cent lead arsenate and 15 per cent hydrated lime. This dust is applied at the time of the falling of the petals, another two weeks later and another one month before the variety is expected to ripen.

Brown rotted fruits must be culled out of the crop as harvested or the decay will spread through the containers and soon spoil the whole lot.

The Black Mold

Hosts. The host range of the black mold is wide. It can grow on nearly any kind of starchy or sugary material. Clara (127) found it growing on the mango in the Philippines. It has been found on strawberries by a number of workers in the U. S. Rose et al (663) reported a study of the fungus on peaches, plums, cherries and other stone fruits in the U. S. Wiant (930) found it a serious disease of melons and cantaloups and Mitra (487) reported it as a disease on jackfruit in India. Lauritzen (363) found the fungus active on snap beans in transit and in the markets when the temperature rose to the neighbourhood of 30 degrees centigrade. He also reported *Rhizopus nigricans* and *R. tritici* as the most generally distributed of the rot fungi causing sweet potato rots. Briton Jones (82) reported that in upper and lower Egypt the black mold caused a severe rotting of the bolls of Indian, American and native varieties of cotton. This was also reported by Neal and Gilbert (541).

The isolation of *Rhizopus* species from the decayed stems of Citrus seedlings which were damping off in nurseries of California has been reported. Koehler (Journ. Agr. Res. LVI, PP 291-307, 1938) found that it was a serious disease on maize seedlings, being especially liable to attack the sccutellum. Nor is the black mould confined to plants but may be found on animals and commercial products Otero (Abst. R. A. M. XV P. 19. 19136) isolated the fungus from the lungs of baby chicks which are suffering from what is called "pulmonary aspergillosis" a disease of the lining of the tubes of the lungs. Schonwald (674) reported the trapping of spores of *R. nigricans* by plates exposed to the air in Seattle, Washington and that when people suffering with asthma were treated with extracts of the fungi 76.7 per cent were definitely helped. Numerous other references to the causing of disease in man and lower animals have been reported in literature. It has also been shown (see under Aspergillus and Pennicillium) that *Rhizopus* species are the cause of much moulding of cotton goods, especially where starchy sizing is used.

Geographic distribution. It appears to be world wide in distribution.

Appearance on the host plant. The most common symptom of this fungus is the dirty black cottony growth which covers the material on which it is growing. This growth consists largely of rhizoids, stolons, sporangiophores and sporangia. Occasionally zygospores may be found. Under conditions of low atmospheric humidity the aerial mycelium may not develop and the substrate is then soft and watery in nature. This is especially true of sweet potatoes stored in a cool dry place. However, there is a characteristic odour of wild rose or of rose geranium which is characteristic of this type of rot and makes identification possible. Under low humidity and temperature the potatoes may mummify and no fungus appear on the surface at all. On sweet potatoes two types of rot may appear; a soft rot which has just been referred to and a ring rot which receives its name from the characteristic manner of rotting a ring around the potato root from the ponit of initiation.

On fruits it usually sporulates profusely and the aerial hyphae are very prominent. On strawberries it causes a heavy exudation of water and the name "leak" is often given to the disease. Other fruits may or may not leak but they generally produce an abundant aerial mycelium.

The organism. Although the term "black mould" may include numerous fungi, the one most commonly intended is *Rhizopus nigricans* Ehr. Butler and Bisby (96) list three species as having been reported in India and Mundkur (517) adds three more. Of this number four are from the soils of India, one from rotting apples, one from fermenting rice and one from *Artocarpus integrifolia. R. arrhizus* has been isolated from the soil as well as rotting apples, hence some of the apparent discrepancy in the total number of reported species.

The aerial mycelium is chocolate coloured at maturity and when sporulating it becomes nearly black. The sporangiophores are unbranched and arise from the nodes of the mycelium one or more at a place. The sporangia are terminal, more or less globose in shape, blackish-olive in colour with a hemspherical columella. The spores are produced in large numbers nearly round in shape, and measuring 11-14 microns.

The zygospores are rounded bodies, measuring 150-200 microns with the epispore covered with warts. In general the zygospores are black. The zygospores germinate by producing sporangia and spores which are the means of initiating the disease after the resting period. These spores germinate by producing a hyphal thread which, on a suitable medium, rapidly develops rhizoids and other sporangia, the rate and amount being proportional to the amount of food material present and available. When mycelia of the proper sex character meet, gametes are formed as previously described, and union is made with the subsequent conjugation and union of nuclei to form the zygospore.

Control. The control of the black mould is closely tied up with the control of storage rots of all kinds. In the case of roots and tubers, dig only when mature and store in a cool dry place.

Avoid bruising and cutting and be sure that they are dry before storing. For fruits and other succulent plant parts, avoid bruising and transfer to a temperature of about 10 degrees centigrade at once after harvesting.

Blue Mould

Host plants. The host range is large. As suggested in the early discussion, the range of substances upon which *Penicillium* may grow is from manufactured commodities to plant products, animals and man. *Penicillium digitatum* and *P. italicum* have been shown to be responsible for much of the loss sustained by the citrus growers due to storage rots. Apples, pears, stone fruits, dates, grapes and many other fruits are attacked.

Geographic distribution. World wide.

Appearance on the host plant. The appearance of the mould on fruit and in culture resembles that of *Aspergillus* but the colour is a distinguishing character. *Penicillium digitatum* is olive green in colour, as compared to the blue of some of the others, e.g. *P. italicum.* Invasion of fruits is usually through a wound, the rot spreading in a circular area around the spot. Under favourable conditions the entire fruit will be destroyed and, as the conidia are produced in enormous numbers, they soon spread to other fruits.

The organism. Penicillum digitatum Sacc. The conidiophores are usually short, being 30-100 microns. The tips of the conidiophores end in slightly swollen cells which cut off successively round conidia. These are in chains and often tangled. The conidia are 4-7 x 6-8 microns.

Sexual reproduction in general is like that of *Aspergillus,* involving a simple oogonium, which may be multinucleate, and slender antheridium which entwines with it in conjugation. Fertilization results in the development of a sclerotial mass within which the asci are developed. The asci are in no regular arrangement but scattered through the tissue.

Control. The discussion of control under the *Aspergilli* will also apply to the *Pencilli* in general. There are, however some differences which make the methods applied to the two genera unlike. For one thing, it has been found that species of *Penicillium* may grow at 0°c. This is important in relation to the storage of the fruit.

Gray Mould

Host range. As noted in the preceding paragraphs the host range of the *Aspergilli* is large. That of *Aspergillus niger* includes fruits, vegetables, animals and men. It is especially bad on onions at times and has been reported to be serious on dates.

Geographic distribution. It is wide spread.

Appearance on the host plant. The diseased portions are covered with a dense mass of gray mould. The colour may be quite dark and for this character some of the mycologists like to put it among the *Dematiaceae.* The tissues decay and dry after which the surface is covered with a mass of conidia.

The organism. *Aspergillus niger* van Tie. It receives its name from the colour of the spores which, as stated above, are dark, nearly black in mass.

The sexual state consists of an ascogonium and an antheridium which come into contact permitting conjugation to take place, after which a sclerotium like mass of tissue develops within which asci are formed.

Control. As the control of the *Aspergillus* in general was not discussed in the preceding paragraphs, it will be included here, since any control measure effective against the common moulds will also be effective against *Aspergillus niger*.

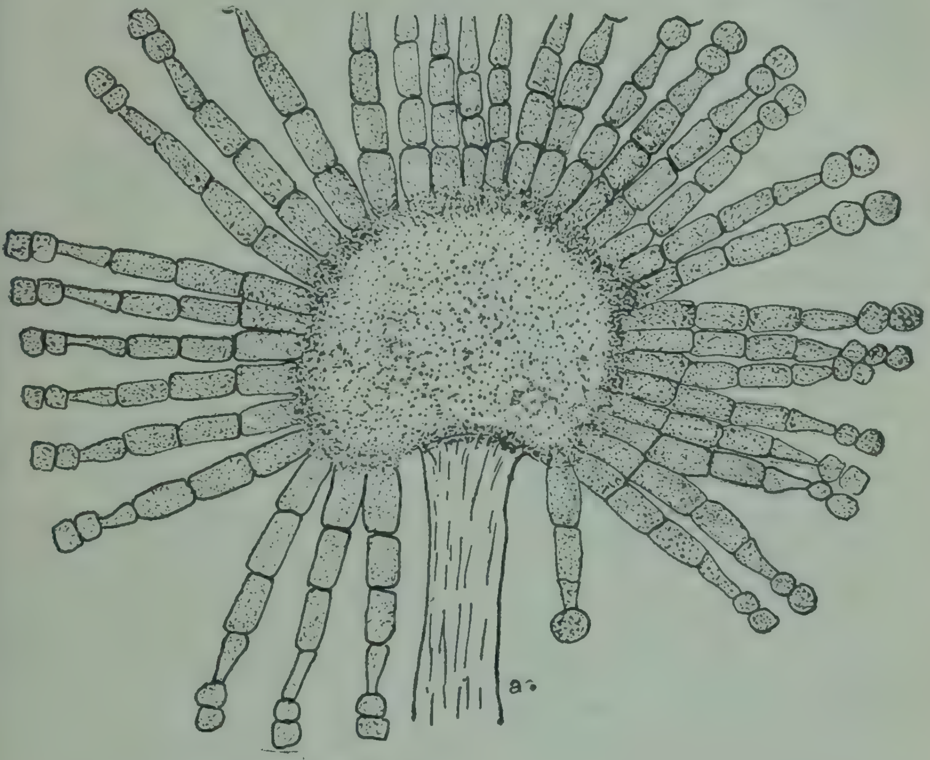


Diagram of conidial head and conidia of *Aspergillus*.

One of the most important things in the prevention of decay by the gray moulds is sanitation. Destruction of mouldy materials is necessary. However, this will not be entirely effective because of the widespread occurrence of the moulds and the ease with which the spores may be borne about on materials and in the air.

Cold storage will reduce the loss and prevent the majority of the moulds from growing. *Aspergillus niger* has been found to be unable to grow below 5°c. (664).

Some of the common Diseases of the Grape in Northern India

There are several diseases found in the grape growing in India but only a few of them are really severe. The downy and powdery mildews are among the more common. Boytrytis rot is also common in some sections.

The Downy Mildew Of Grapes

Hosts. Most of the species of *Vitis* are affected by the fungus. This is especially true of the cultivated *Vitis vinifera.*

Geographic distribution. It is present in nearly all parts of the world where grapes are grown. Uppal (843) and Venkatarayen (890) reported it in the Bombay presidency on grapes.

Appearance on the host plant. The disease appears first as patches of light green on the upper surface of the leaves. On the lower side of these patches the fungus develops among the leaf hairs and is less conspicuous. But if the atmospheric conditions are optimum there is developed a dense mass of conidiophores and conidia which give the appearance that has given rise to the name "downy mildew".

As the spots become older they become brownish and are more conspicuous. If the air is dry the growth of conidia is scanty and the spots likely to be smaller. Under favourable conditions the whole of the leaf may be involved.

On the shoots, the tip may appear watersoaked and there will be a dwarfing of the tender portion. There may be some swelling of the tip and the leaves remain small and curled. Upon close examination the diseased portion will be found to be covered with the downy growth of branched conidiophores and conidia.

Flowers and fruits may be attacked and the flowers killed. Fruits become a grayish lead colour and the surface hardens so that the berry becomes shriveled and small. These may become mummy-like in appearance and remain on the pedicel for long periods after the other fruit has fallen.

The organism. The fungus of the grape mildew is known as *Plasmopara viticola* (B. and C.) Berl. and de T. It was first called *Botrytis viticola* by Berkely and Curtis and then the true relationship to the *Peronosporaceae* was shown when De Bary called it *Peronospora viticola.* Berlese and De Toni described the fungus as *Plasmopara* in 1888.

The vegetative mycelium is typically coenocytic, developing within the intercellular spaces as thin-walled, irregular hypha. The haustoria are knob shaped, penetrating the cells. The hyphae are very irregular in size. In some cases the hyphae are as much as 40-60 microns thick where the tissue is loose and there are large intercellular spaces.

The conidiophores are large and irregularly branched, the branches coming off from the main one at right angles with each small branch giving off branches at nearly right angles. This makes the whole conidiophore appear like a tree of crosses. The conidia (sporangia) are borne on short finger-like stalks at the tips of the branches. The conidia are egg-shaped or slightly elliptical, 9-12×12-30 microns. They germinate to produce swarm spores. If a swarm spore is able to find its way to a leaf it soon loses its cilia and then germinates to form an infection thread that enters the first stoma it finds and starts an infection. Within 5 to 20 days after infection the characteristic spot appears on the leaf, shortly the conidiophores emerge through the stomata and the new crop of conidia are ready to be distributed.

Antheridia and oogonia are produced within the leaf and the oospores are soon found. There is a difference in the germination of the oospores as compared to some of the others. The oospore of *Plasmopara* germinates by sending up a germ tube at the top of which is a single conidium. This conidium produces swarm spores in the same manner as for the vegetative conidia described above. All that is necessary for infection is the placing of one of the conidia, or the zoospores, on a vine leaf.

Life cycle. The early season infections will come from the dormant mycelium in the diseased tips of the twigs, or from the oospores in the diseased leaves on the ground. Infections thus initiated are carried on throughout the season by the conidia formed on the diseased areas of fruit, twig and leaf. As the season draws to a close oogonia and antheridia are formed and oospores are formed from the union of gametes. These carry the disease over the dry or cold season and the next growing season the same cycle is repeated.

Control. Sanitation and spraying, together with resistant varieties, form the best way of control. Sanitation consists in removing all of the diseased leaves and twigs from vineyard. If the prunings are all removed from the vineyard and burned there is much less material for the following season's infection.

Spraying with Bordeaux is one of the most common methods of control. Various strengths of spray have been recommended. Bombay Leaflet No. 8, 1931, gives the following recommendations for the control of the diseases of the grape.

1. Apply Bordeaux mixture 5-5-50 when the shoots are 6-8 inches long.
2. Apply sulphur dust at the rate of 20 ℔s. per acre within 2-3 days after Bordeaux mixture is dried on the leaves.
3. The second application of sulphur dust should be made at the time of blossoming; use 30 ℔s. per acre.
4. The third application of sulphur dust should be made about 40 days after the second dusting; use 30 ℔s. per acre.

Venkatarayan (890) states that the application of three sprays of Bordeaux during September and October, costing only about 1 anna per plant per application, will control the downy mildew.

There are several varieties of grapes in the various grape growing regions which have shown some resistance. The grower would do well to consult his nurseryman, mycologist or successful neighbour before planting, in order to learn the best varieties for his section.

The Powdery Mildew of the Grape.

Hosts. The members of the genus *Vitis.* In India it has been reported on *Vitis vinifera.*

Geographic distribution. Kashmir and Poona at least. Throughout the world in the area where the grape is grown.

Appearance on the host plant. The vegetative mycelium is superficial but because of the haustiria it is persistent on the leaves. The conidiophores are short, septate and bear the conidia in short chains of two or three. The mass makes a white powdery appearance on the leaves so that it is easy to differentiate. As the mycelium grows older the mass may change to a brown and thus the leaves become browned, especially on the under surface. Some distortion may occur.

The organism. Uncinula nectator (Schw.) Burr.

The conidia are elliptic oblong to rounded, in short chains on short conidiophores. They are hyaline and measure 25-30 x 10-12 microns. The mycelium is thin walled with the haustoria arising from swellings on the threads. These penetrate the epidermis with a slender projection that is filament like and after entering the host cell body it swells into a bladder like structure. These, together with the host cells, die and turn brown. Thus the browning of the leaves referred to above.

Ascocarps are found over the surface of the leaves and form after the appearance of cool weather. The ascocarps are brown when mature and have long appendages which curl at the tips. In some countries they are not found regularly.

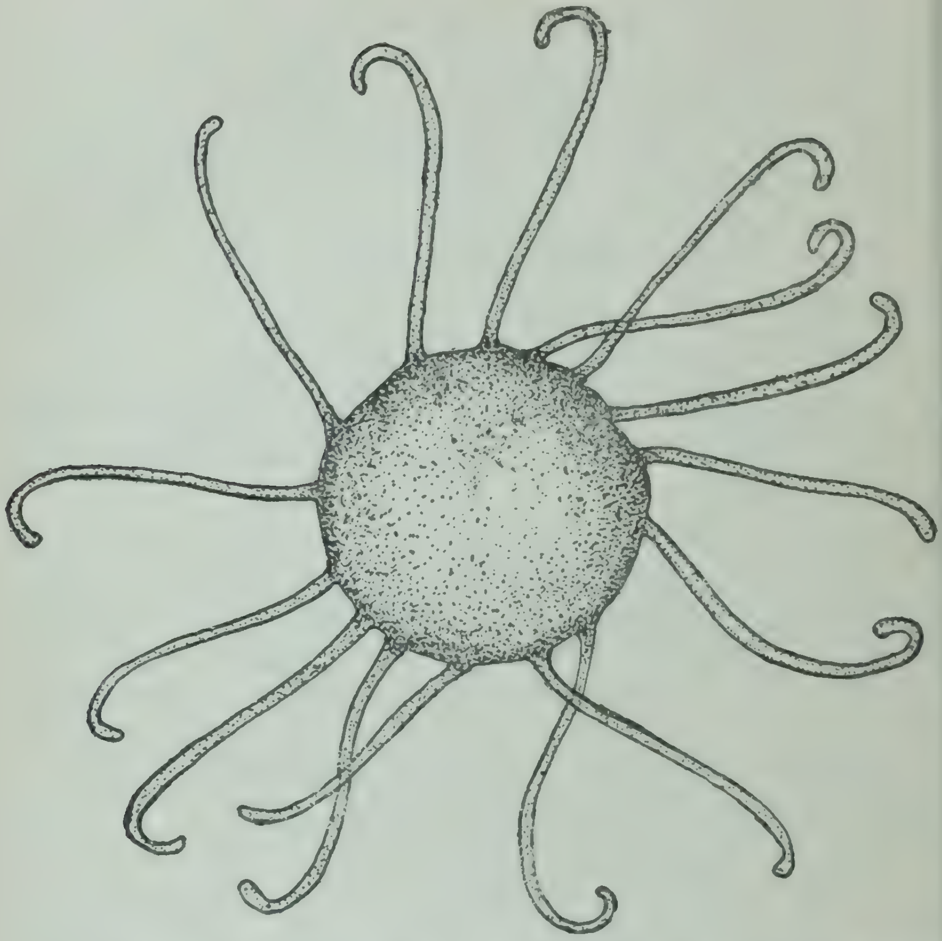


Diagram illustrating the ascocarp of powdery mildew of the grape, *Uncinula necator*.

Control. Bordeaux mixture was one of the first to be used for the control of powdery mildew. More recently the application of sulphur dust has been the recommended control. Rose et. al. (664) state that one to six application of the dust will usually give satisfactory control. They state that the sulphuring should be begun in the early season of growth so that the first is when the canes are 6-8 inches high, the second when they are 12-18 inches and third when 2-3 feet long. Later as the season may develop favourable for the fungus.

Gray Mould Grapes

Geographic distribution. Botrytis species causing rot of soft fruits, have been reported from numerous sections of the world. Rose (664) gives an account of the disease caused by the fungus in the United States. Asghar (I. J. A. S. IX, 719-727, 1939) reported the disease on grapes in the Quetta Valley.

Appearance on the host plant. Botrytis rot is frequently called the gray mould because of the olivaceous growth of mycelium. Berries turn soft and a rather pleasant sweet odour comes from them. The infected grapes usually remain firm and there is no marked collapse of the tissue. The fungus spreads from berry to berry and there is usually a nest of diseased fruit where it occurs. If the bunch is shaken the berries will shatter and fall but they will leave a small portion of the fruit still attached to the stem. Usually the berries that fall will have a brownish cast when held up to the light. This condition is likely to result after rains. Asghar (28) states that cracking may occur before rotting. Healthy berries may be attacked but bruises and cracks aid. Humid conditions aid the spread of infection.

The organism. The organism is known as *Botrytis vulgaris* Fr. There are other species of *Botrytis* that have been described as causing a similar rot of fruit. Among the more common is the *Botrytis cinerea* which is responsible for a rot of Citrus fruits.

The conidiophores of *B. vulgaris* are large, erect, olivaceous, constricted and several millimeters high. They may be simple or branched. The main branches are 10-25 microns thick. Secondary branches are 7-9 microns in diameter. Conidia are sub-hyaline, granular, appear grayish-olivaceous in clusters.

Sclerotia are formed abundantly in culture. They are formed by the hyphae twisting about each other. Young sclerotia are whitish at first but later become dirty greenish and then dark brown to black. These sclerotia form secondary conidia over the surface. The optimum temperature for growth was determined as 27°C. but the best temperature for sporulation was 23° C. A temperature of 30° inhibited the growth.

Control. Temperature and humidity are the two most important factors in the development of an epiphytotic of gray mould. In the field, pruning and trellising to permit a rapid drying during the damp foggy weather will aid in the control. In storage it has been found that sulphur dioxide fumigation will control the spread of the fungus.

SOME DISEASES OF MISCELLANEOUS FRUITS OF NORTHERN INDIA

There are a number of fruits that may be classed as miscellaneous from the stand point of diseases because they are not attacked by many serious disease producing fungi. Some of them will be mentioned in the following pages.

Ceratostomella Disease of Pineapple

Host. The pineapple (*Annanas comosus* Merr).

Geographic distribution. The pineapple growing regions of India and the Malayan area.

Appearance on the host plant. The leaf spots appear on any portion of the leaf. They vary considerably in shape, size and colour. Some are large and white and may be seen from a considerable distance, while others are small and inconspicuous. The typical spots have a straw coloured central portion with a dark margin. Dark areas within the straw coloured spots are caused by the formation of macrospores. The early diseased tissue soon dries out, shrinks and leaves the portion with a twisted or distorted appearance. The spots vary from olive brown to white and from regular to irregular in shape.

Base rot. Pineapple suckers, after planting, develop normally for a time and then become inactive, develop a yellowing of the leaves, with withering and then drying of the suckers. Such suckers will be loose in the ground and will have black areas at the base of the stem where the fungus has invaded the tissues. The rot may extend until the whole of the sucker, as well as the leaf base, may be invaded. The plant may break off at the ground level. Sometimes older plants are also affected.

Fruit rot. On the fruit water soaked areas, yellowish at first, becoming darker in colour, may form. There is a characteristic odour which accompanies this rot. The diseased tissue is soft to the touch and becomes black when exposed to the air. The black colour is due to the mass of macrospores which form on the surface. These will appear after some 24 hours exposure to the air. In advanced stages the whole fruit becomes covered with spores.

The Fungus Ceratostomella sp. The mycelium is hyaline, granular, may be greenish with numerous branching and septations. They vary from 2·5—10·5 microns.

Conidiophores are of two types, macro and microconidiophores. The former are clubshaped, septate, hyaline, occasionally granular, with 1-3 septations. They measure from 21·9-73·2 microns. Microconidiophores are swollen at the base and taper to the point.

Macroconidia are light to dark brown, smooth, ovoid or elliptical with thick walls. They are formed in chains of as many as 30 spores. In size they range from 14-19×10-14 microns.

Microconidia are of two types. One is light brown and the other hyaline. The light brown conidia are barrel shaped with thick walls. They measure 9·4-19×4·6 microns and occur in chains of as many as 50. The hyaline microconidia are cylindrical with thin walls.

They measure 9-12×3·5-5 microns and occur in chains of 2-75.

Control. None suggested at present except rotation and destruction of diseased plant parts.

Wilt of Pineapple

Hosts: The organism is wide-spread causing root rot of numerous plants.

Geographic distribution. Wide-spread as far as the fungus is concerned but the disease has been reported only in Assam in this country.

Appearance on the host plants. According to Chowdhury (120) the first symptoms are a yellowing of the leaves of two year old plants. The leaves actually becoming an olive drab colour, more or less collapse and droop. This is the most striking symptom of the disease. Later the leaves shrivel from the tip downward.

Fruit development is arrested and there is early colouring, a spongy texture and an insipid taste.

The stalks remain erect and the withered fruits are hard to dislodge. The roots may be the first attacked and be decayed before any other portion shows the disease.

The organism. *Phytophthora parasitica* Dastur.
(*See under Foot-rot of Pan*).

Control: At present there is no control suggested. The habit of growing the pineapples among the trees on the hillsides of Assam, where the disease has been first reported, makes control of a soil borne organism a problem.

Leaf Spot of Jack Fruit

Hosts. It is found on a wide range of hosts. The list includes many of the *Cucurbitaceae*, papaya fig, and others.

Geographic distribution. World wide.

Appearance on the host plant. The spots appear on the leaves as large, circular, dark-coloured areas with rather sharply delimited margins. The colour of the diseased areas is usually dark brown to black but there may be a reddish shade over portions. The areas vary from 10-30 mm. with up to 10-15 spots on a leaf. If the leaf is badly infected it will fall. See photograph.

The organism. Colletotrichum lagenarium (Pass.) Ell. and Holst.

For the description of the fungus see under Anthracnose of Papaya.

Control. No control measures have been worked out for the disease as yet. Destruction of the diseased leaves is evidently one thing that will aid in reducing the attack. Bordeaux or Bergundy mixture may also be of value.

Physalospora Disease of Guava

Hosts. Guava is the only one reported.

Geographic distribution. Reported by Uppal (850) in south India.

Appearance on the host plant. The first appearance is on the bark of the branches and the spread is rapidly from one branch to another. The wood dries, cracks, the bark cracks off and the branch dies. If the disease spreads very widely over the tree the whole tree dies.

The organism. Physalospora psidii Stevens and Pierce.

Stevens and Pierce (743) state that the mycelium is inter-cellular, brown, continuous, hyaline. The microconidia are elliptical hyaline, 5-7×2-3 microns and the macroconidia are 12-15×5-8 microns. The conidiophores are 10-13 microns long.

The perthecia are scattered, sunken in the bark, subglobose, short protruding ostioles but with a beak that may be 120-165 microns high. The diameter of the perthecia is between 270 and 345 microns. The asci are club shaped (clavate), tapering towards the ends, especially the base and typically 8-spored. The asci measure 70-100×26-33 microns. Ascospores are elliptic and measure 30-37×13-16 microns. Uppal (850) reported that the perithecia were found over the bark on the dead branches.

Control. So far no control measures have been found for the disease. It has not appeared in the United Provinces. At least it has not been reported so far.

Wilt Disease of Guava

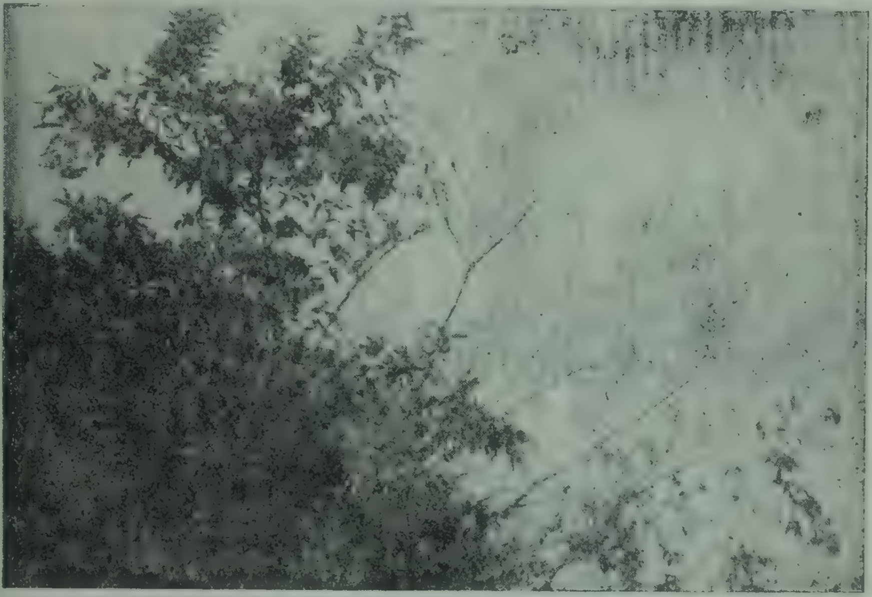
Hosts. At this time only guava has been reported.

Geographical distribution. It has been reported over a considerable portion of the United Provinces.

Appearance on the host plant. The first symptoms are usually a wilting and browning of the leaves. These usually do not fall for some time. The bark will show slightly darker coloration on the outside. An incision will show a darker coloration down to the cambium layer. It is not usually discoloured beneath the cambium on those parts recently infected. Infections extend up and down the stem in streaks which may not girdle the stem and thus it is not uncommon to find a limb with part of the branches showing dead or dying leaves and the rest of it still green and apparently healthy.

After a time the leaves fall and the stems and branches are bare and dark coloured. The disease spreads from branch to branch apparently through direct contact. Just how it is carried from tree to tree is not known.

The first instance of the disease being noted in the Allahabad area was in 1939. By 1941 it was becoming severe in several orchards and was probably present for some seasons but went undetected as a disease. Isolations from a small orchard along the Rewa State road some two miles from the Agricultural Institute yielded a fungus which was identified by the Division of Mycology, Indian Agricultural Research Institute, Delhi, as a species of *Cephalosporium.* The departure of the Plant Pathologist from the Institute at that time interrupted the study of the disease.



Photograph showing the appearance of the sky line of a guava tree affected with the wilt disease. Photo taken at Allahabad.

The organism. At present it is not certain but Das Gupta and Rai (Current Science VI, pp. 256-258, 1947) have isolated a species of Fusarium from diseased trees. As the microspore stage of the Fusarium is often referred to as *Cephalosporium* it is possible that the fungus isolated at the Agricultural Institute in 1941 and the one found by Das Gupta and Rai, referred to above, are one and the same fungus.

Control. The fungus gives promise of being one of the worst diseases of guava to have appeared in India. At this time there is no recommended control measure. Prune out and destroy the prunings. It is about the only measure that we now know to suggest.

CHAPTER XIII

SOME OF THE MORE COMMON DISEASES OF PAN ((PIPER BETLE) AND TEA (CAMELLIA THEA)

Pan (*Piper betle*) is one of the most important crops grown in certain regions of Northern India in Bengal and especially the Khasi Hills region of Assam. There it is one of the main income crops of the Hill people. It is cultivated in the forest on the steep hillsides and in many cases the vines are permitted to climb on to the native forest trees, which, in Assam, may be jack fruit or other trees of commercial value.

Perhaps the most important single fungus causing disease on the pan plants is *Phytophthora parasitica.* It is closely associated with two rather distinct diseases and yet they are associated as they are largely the result of being parasitized by one fungus. Foot rot and leaf and stem spot are caused largely by the one fungus species. There is also a bacterial leaf sot which sometimes becomes serious.

Foot-Rot Disease of Pan (Piper Betle L.)

Hosts. In India, *piper betle, Ricinus communis* and *Colocasia antiquorum* are attacked by several different fungi. Dastur (159) and McRae (447) consider that the *Phytophthora* on *piper betle* is distinct from *Phytophthora colocasia* reported on *pan* from Malaya by *Thompson* (813) and identified by Ashby (34). It is evident that there is much to be done on the foot-rot diseases and that we may expect further reports.

Appearance on the host plant. The disease appears to have been recognized on *pan* for at least 80 years. It was epidemic in some seasons and from reports must have destroyed the entire crop of some villages. When this happened year after year the cultivation of *pan* was discontinued for some time.

The first symptom of the disease is a darkening of the stem which may be preceded by a loss of colour in the leaves and a drooping of the tender portions. Examination of plants in this stage will show that they have a rotted root system and the stem will usually break at the surface of the ground. By the time the plant dies some two or three nodes and internodes will be involved in the diseased area.

As the disease progresses the diseased portions may rupture and the tender tissues which are exposed, shrink, become slimy and slough off. All that is left will be the vascular tissue fibres. Mcrae (447) reports that if the plant is cut off just above the diseased area and placed in water it will recover and this indicates that the disease is confined to the discoloured area.

The first evidence of the disease in the roots will be a dying of the smaller roots. It appears to travel up the stem by way of the vascular bundles which are blackened. Only one bundle may be invaded or many may become diseased. The number is reflected in the rapidity of the disease development and the extent of the discoloration. When the infection is near the soil line the plant dies much sooner than if the invasion is farther up the stem.

The practice of hilling up the plants as they grow from season to season may make it possible for roots to develop from some of the upper nodes and thus the true condition of the root system is hidden until the tops begin to show the typical symptoms. For this reason the disease may appear to develop suddenly but this is hardly likely under most conditions.

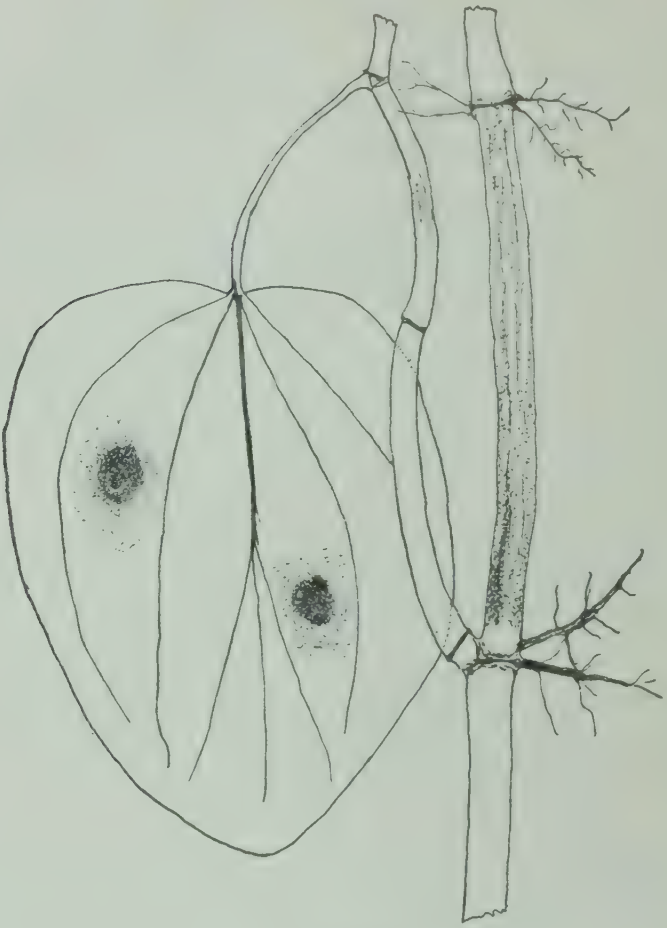


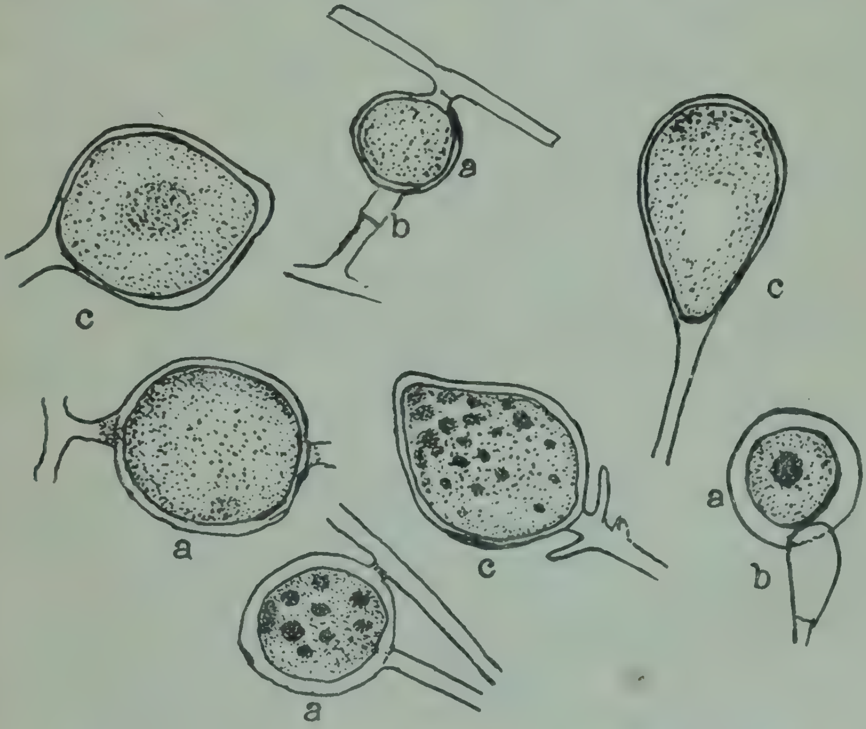
Diagram of Pan (Piper betle) stem and leaf showing infection by *Phytophthora parasitica.* Specimen collected near Laitkynsew, Khasi Hills, Assam.

The organism. McRae (447) found that at least three organisms were associated with the disease in Bengal: *Phythophthora parasitica* Dastur, *Rhizoctonia solani* Kuhn; *Sclerotium rolfsii* Sacc. Dastur (159) reported the following as associated with the disease in the Central Provinces: *Phytophthora parasitica* var. *Piperina* n. sp.; and *Pythium piperinum*, nov. sp. The attack of the fungus varies with the species of fungus.

McRae (447) observed that the attack of *Rhizoctonia solani* did not make headway until the cooler part of the season, while the warmer portion of the season found *Sclerotium rolfsii* and *Phytophthora parasitica* active. He noted that much of the loss in Bengal is due to the *Phytophthora*. In the Central Provinces, Dastur (159) finds that *Pythium* spp. cause an amount of damage more comparable to that of *P. parasitica.*

According to Dastur, *Sclerotium rolfii* is probably not an active parasite but more likely a faculative saprophyte.

Dastur (159) gives the measurements of the hypha of *Pythium piperina* as 2.2 to 5.75 microns. They branch freely and may form the typical knotted bodies



Diagrams of reproductive structures of *Phytophthora parasitica* grown on oat meal agar. Culture kindly furnished by Dr. B. B. Mundkur.

a. Oogonia. b. Antheridia. c. Sporangia.

of the *Pythiaceae*. Conidia measure from 12.5-20.4 microns with the average from 15.3-20.3 microns. Zoospores from 3.4-5.1 microns with the cilia approximately twice the length of the spore.

Oogonia are globoid or spherical and either lateral on the hyphae, terminal or intercalary (within the mycelium). They are smooth-walled and hyaline and measure from 15·3-25·5 microns. Antheridia may be only one or many to each sporangium. Oospores measure 12·75-20·4 microns.

McRae (447) measured the sporangia of *Phytophthore parasitica* which he isolated in Bengal and found that the mean length of 400 sporangia was 40.03±.28 and the mean breadth was 17.62±19 microns. Dastur considered his isolation as a variety of *Phytophthora parasitica* while McRae made no such claim for the isolations made in Bengal.

The disease caused by *Sclerotium rolfsii* differs from the preceding in that there is a distinct white feathery growth of mycelium over the outer surface of the tissues under the soil. The sclerotia appear at first as feathery white bodies that soon change to darker coloured, hard, mostly smooth surfaced bodies that are more or less brownish in colour. Dastur gives the diameter of the sclerotia as from one to one and a half millimeters.

McRae, studying the measurements of the *Rhizoctonia* strain isolated and comparing with measurements given by Duggar and others, concluded that it was *Rhizoctonia solani.*

Pythium, *Phytophthora* and *Sclerotium* begin attack with the opening of the rainy season in the various sections. In the case of *Pythium* and *Phytophthora*, sporangia form soon after the attack begins and the zoospores are then a means of spreading the disease. Water is the medium by which the zoospores are distributed. In the case of *Sclerotium* and *Rhizoc-*

tonia, the diseases are probably spread by the sclerotia which may also be carried in water.

Control. During the rainy season the weeds grow rankly and offer ideal conditions for the fungus, if left to grow about the vines. Clean cultivation has been found to help. McRae (447) mentions the use of Bordeaux mixture as a spray and also for soaking the cuttings before planting. Dastur (159) reports the use of Bordeaux 2-2-50 to irrigate the lines and also to spray the plants and that the result was a saving of practically 100% of the plants. One difficulty in controlling the diseases is that they are probably living on a number of other host plants about the fields. This fact makes sanitation all the more important.

Control of the *Rhizoctonia* in Bengal was tried with a compound known as Kerol and some encouraging results were obtained. But Dastur makes the comment that the patent compounds are too expensive and that Bordeaux is just as effective and cheaper. In that case it would seem that his suggestion of irrigation with Bordeaux 2-2-50 should control the four fungi under the soil and using the same strength for a spray should be effective on the leaves in the case of *Phytophthora.*

Phytophthora Leaf Spot and Stem Rot of Pan

Hosts. The fungus has a rather wide host range and has been found widely distributed both in this country and in other parts of the world. It has been found on *Piper betle, Ricinus communis, Rheum officinale, Colocasia antiquorum, Citrus* species causing gummosis (see under Gummosis of *Citrus*) and many other hosts.

Geographic distribution. Widely distributed.

Appearance on the host plant. On the leaves the spots appear as more or less irregular rounded areas that are first yellowed and then brown. There may be more or less defined concentric rings of light and dark coloured tissue. The centres may fall out as the disease progresses. If many spots occur the leaves are likely to die and fall or the infection may also occur on the stem. When stem infection occurs the whole plant may die from the point of infection upwards. Stem infections are marked by a darkening of the portion at the point of infection. The infected area may extend for several inches along the stem or it may be only a short distance.

The organism. *Phytophthora parasitica* Dastur. There appears but one fungus that has been held responsible for the leaf spot and stem rot of pan. Since *P. parasitica* is also one of the foot rot causing organisms, it would be logical to suppose that the two types of disease would be present in the same plantings. However, in the Khasi Hills, Assam in 1947, where leaf spot was observed, no foot rot was seen. Inquiry of the growers there failed to elicit information that would verify the suggestion made above although there was considerable leaf spot.

The fungus consists of a much branched, non-septate mycelium which is hyaline. The conidiophores are branched, emerge from the stomata singly or in groups and bear irregular thickenings below the apparently lateral conidia.

The conidia are at first terminal, but with progressive growth of the hyphal thread, are pushed to one side so that they appear later on. They are lemon shaped with a papilate point which swells and then ruptures to permit the zoospores to escape.

The oogonia are commonly produced on corn meal but the character is apparently not a reliable identification according to Tucker (823). Oospores are spherical, thick walled and the antheridium will be usually found clasping the stalk. See diagram under

Root Rot. Chlamydospores are also found in most isolations.

Life cycle. The life cycle is not difficult to trace. The fungus can apparently live over as oospores in old diseased leaves and stems. Following germination of oospores, infection may take place on young leaf or stem. Such infections will produce conidiophores and conidia and these may be carried by wind, water, insects or birds to other parts of the same plant or different plants.

Chlamydospore germination results in the production of mycelium directly and then conidiophores and conidia. These then carry on the infections for the rest of the growing season.

Control. Dastur suggested Bordeaux mixture for the control of the foot rot and it is likely that the same treatment will help in the control of leaf and stem rot as well. In the Khasi Hills of Assam the growers say that if they remove the first infected leaves and destroy them there may be no more infection. Infections on the stem are scraped and, if the scraping is done with a sharp knife and all discoloured tissue is removed, the stem may recover. If the infection occurs in the upper leaves of a plant there is likelihood that the infection will spread over the whole plant. If the lower leaves are removed early in the season they believe that they may prevent infection. If a stem has been infected and it is cut below the infection, new growth will occur and often no further infection will occur. These are the methods suggested by those who grow the pan in the forests. It is held that pan and jack fruit are especially companionable as crop plants and the *Piper betle* plant may be seen growing on the jack fruit trees to a height of 30 to 40 feet and the gathering of the leaves necessitates climbing to these heights.

Bacterial leaf spot of Piper Betle

Hosts. Piper betle.

Geographic distribution. Ceylon, Burma and India.

Appearance on the host plant. The first symptoms are minute water soaked spots on the under surface of the leaves. Later these appear on the upper surface also as dark, rounded or angular areas surrounded by a yellow zone. On the under surface these zones have a water soaked appearance. The centers of the spots are mottled brown, later turning black and rotting, even falling out. When badly infected the leaves turn yellow and fall. When the weather is extremely humid there may be an exudate form on the lower surface. Stems may be infected and if this is so the plants usually die.

The organism. Bacterium betle Raghunathan.

Non motile; 0·5-2·5 microns, short chains; no spores are produced, no capsules; aerobic; gram negative.

Control: Diseased leaves should be destroyed immediately upon identification. General sanitation.

The measures recommended are 2:2:50 Bordeaux every two months. The spray will control *Colletotrichum, Gloeosporium* and the leaf spot caused by *Phytophthora parasitica var. piperina Dast.* Bordeaux 4:4:50 will control bacterial spot as well as the foot rot.

Some of the More Common Diseases of Tea (*Camellia thea*)

Tea is an important crop in a number of sections of India and, as is true with other crops which are grown intensively in some areas, there are a number of diseases which attack it. Canker, *Cercospora* leaf spot, red rust (*Cephaleuros mycoides*), root rot, copper blight, brown blight, gray blight and others will be briefly discussed in the following pages.

Tea Canker

Host. This is known in India as canker when it is found on trees. Butler and Bisby (96) report it on species of *Populus, Prunus* and *Pyrus* as well as on *Camellia thea.* In the United States, it has been found on a number of important forest trees (Dodge and Rickette: Diseases and Pests of Ornamental Plants. Jaques Cattel (1943).

Geographic distribution. It has been reported from nearly every country in the world and from many groups of plants. It has been found associated with the orchards of apple and pear in the United States and Europe. In India it is mostly associated with tea plantations.

Appearance on the host plant. Tunstall (826) has made numerous observations on the *Nectria* which is found on tea in the Surma Valley of northern India. It begins on the small twigs and spreads to the larger ones causing a die-back. But his observations were that it could not establish itself on tea as an active parasite on healthy tissue (826) without either a wound or dead twig to act as a passage way for the mycelium. It may produce galls on the side of the branches and in the case of small twigs may girdle so that death soon results. In some cases the fungus does not produce definite galls but beneath the surface masses of hyphae grow and then rupture the epidermis and become exposed as stromatic masses.

The organism. The organism is known as *Nectria cinnabarina* (Tode) Fr. The mycelium is closely septate and grows over, or into, large areas of the vascular tissues beneath the epidermis. It appears to confine itself largely to the cambium and soft bast region of the stem.

The conidial stage appears during the growing season of the host and conidia are found in large numbers on the stromatic masses that break through the epidermis. The conidia are of two types, one being the large macroconidia, usually *Fusarium*-like, and the small microconidia, like the spores of the Imperfect genus *Cephalosporium* i.e., they are from 1 to 3-celled but do not have the familiar curved shape of the larger macrospores of *Fusarium.*

Following the conidial stage, the perithecial stage develops on the host tissue. The stromatic mass develops within its tissue a globular perithecium which consists of a wall of interwoven hyphae and within this the asci are borne. They develop from the base and converge towards the apex or ostiole. The asci are club-shaped and measure 60-90×8-12 microns. The spores are two-celled but the cells are unequal. There are typically eight ascospores measuring 14-16×5-7 microns.

Control. The fungus over winters in the diseased wood and its period of activity is corelated with that of the growth of the host. It may be spread by ascospores or conidia but they are not able to penetrate the healthy wood so must enter by way of wounds or dead branches. Thus the most effective control is to prune all of the dead branches out of the tree and, if the cuts are large, to paint the cut surfaces with a disinfectant. Tunstall (826) recommends lime sulphur for the cut surface. In the case of tea, he recommends that the cuts be made close to healthy wood in order that there be as little diseased wood as possible.

Leaf Spot of Tea

Host plant. It appears to be confined to the tea plant.

Geographic distribution. It has been reported in the tea-growing sections of South India and it is considered that this is the same species that is common in Ceylon.

Appearance on the host plant. The fungus causes a spoting of the leaves, with small circular diseased areas that appear towards the end of the season.

The organism. Cercosporella theae Petch. The fungus produces its conidiophores in clusters on the diseased portion of the plant. It is more serious in damp misty weather. The mycelium is hyaline throughout. The conidiophores emerge through the stomata in groups. Conidiophores, mostly simple with conidial scars prominent. Conidia filiform, one to many septate, hyaline.

Control. Sanitation. Bordeaux spray when serious.

Red Rust of Tea

Hosts: Camellia thea.

Geographic distribution. Common in the north east of India but less so in the north west.

Appearance on the host plants. The disease appears on the upper surface of the leaves as reddish brown disc-like spots. The spots appear only on the leaves and have not been observed on the stems. The disc-like structures are attached to the upper surface although there are no hyphae that penetrate the epidermis.

The organism. Cephaleuros mycoidea Karst.

Sporangiophores are borne as erect, orange-coloured filaments. These are formed during moist weather. Each sporangiophore bears a number of sporangia which break off and form zoospores which are capable of setting up a new area of rust.

Control. As the algae is a surface inhabiting plant it does not directly injure the host. Its main damage is in the interference with photosynthesis.

Root Rot of Tea

Hosts: Thea chinensis. Saccharum officinarum, Hevea brasiliensis. Cinchona spp.

Geographic distribution. North-east India, Assam, Burma.

Appearance on the host plant. The diseased plants either do not produce any new shoots after pruning or only a few leaves that may become mottled with patches of yellow, later turning black and dying off. No external hyphae can be seen on the diseased roots but sections will disclose the presence of dark brown, almost black hyphae with thin walls.

The organism. Botryodiplodia theobromae Pat.

The fruiting takes place on the dead twigs where pycnidia are found. The hyphae are hyaline at first, later turning black. In culture the hyphae often fuse to form a net like structure. The pycnidia are globose or flask shaped, black, carbonaceous and have distinct outer and inner walls. They measure 280·6-561·2 microns. The conidiophores are hyaline and continuous when young but later become one-septate and dark coloured. The pycnospores measure 19.2-25.6×7.2-11·2 microns.

Control. Root rots are hard to control. Rotation and organic manures are probably the best means of control.

Copper Blight of Tea

Hosts: Camellia thea.

Geographic distribution. Recorded by Petch in the Kangra Valley.

Appearance on the host plant. The symptoms resemble those of Brown spot (*Phoma theicola*) in general appearance. They may enlarge and extend to the leaf margins. On the upper surface the spots are at first yellowish brown, later become copper coloured and finally gray. In the latter respect they resemble gray blight. The spots change from circular to irregular in shape. When fully aged the spots show a sharp demarcation between the dead and the healthy tissue. On the under surface the spots show a grayish brown colour. Minute black perithecia form in the diseased portions. The epidermal cells of the diseased portion break and this causes a characteristic appearance.

The organism. Guignardia camelliae (Cooke) Butler.

In culture the perithecia are 109-274 microns with the measurements of those found on the host somewhat smaller.

Asci are elongate, elliptical and typically 8-spored. They measure some 60 microns in length. Ascospores are elliptic, hyaline and continuous in two rows. They measure 17·6-20·8×5·6-7·2 microns.

Control. Destruction of the diseased material. Although not suggested by any of the workers it would seem that where the disease is severe a copper spray could be used with advantage.

Brown Blight of Tea

Hosts: Camellia thea.

Geographic distribution. In India.

Appearance on the host plants. The first symptoms are a yellowing of the normal colour in local areas. As the spots become older they become dark brown and ultimately turn a chocolate brown. They may turn almost black. One of the characters that may be used in identification is a yellow band about the spot caused by the mycelium advancing into the healthy tissue outside the brown area. Acervuli are found mostly on the under surface of the leaves. There appears a tendency for these to group along theveins.

The organism. Colletotrichum camelliae Massee.

The acervuli are pinkish with dark setae that are scattered around the margins. The setae are generally few, dark brown, bristle like, may be slightly curved and measure 33-83×4 microns.

The conidia are hyaline, 10-21×3·5-5 microns and are borne on erect conidiophores.

The perfect stage is a *Glomerella* which has been obtained on artificial media. Perithecia are pear shaped with a short neck. They measure some 126 microns. Asci are borne sessile, are hyaline, and measure 72 microns for the average length. The ascospores are typically eight, hyaline, slightly curved and 12·6-16·2× 3·4 microns.

Control. Destruction of infected leaves appears to be one control. It appears to be subject to the weather and not usually serious.

Brown Spot of Tea

Host: *Camellia thea.*

Geographic distribution. Petch (605) reported it in the Kangra Valley.

Appearance on the host plant. The first symptoms are reddish brown patches on both surfaces of the leaf on which minute dark coloured pycnidia are borne. The infected spots disintegrate and break in many places. Hyphae are profusely seen in sections of the infected spots. The symptoms are similar to the Brown blight but differs in the fruiting body which is a definite pycnidium.

The organism. Phoma theicola Petch.

The pycnidia are globose, measuring from 96-300 microns in diameter and broadly oval. Spores are hyaline, non-septate, biguttulate, measuring 3·2-12·8 ×1·6-7·8 microns. A definite ostiole is found in the pycnidium.

Control. No control measures are given for the disease at present.

Gray Blight of Tea

Host: *Camellia thea.*

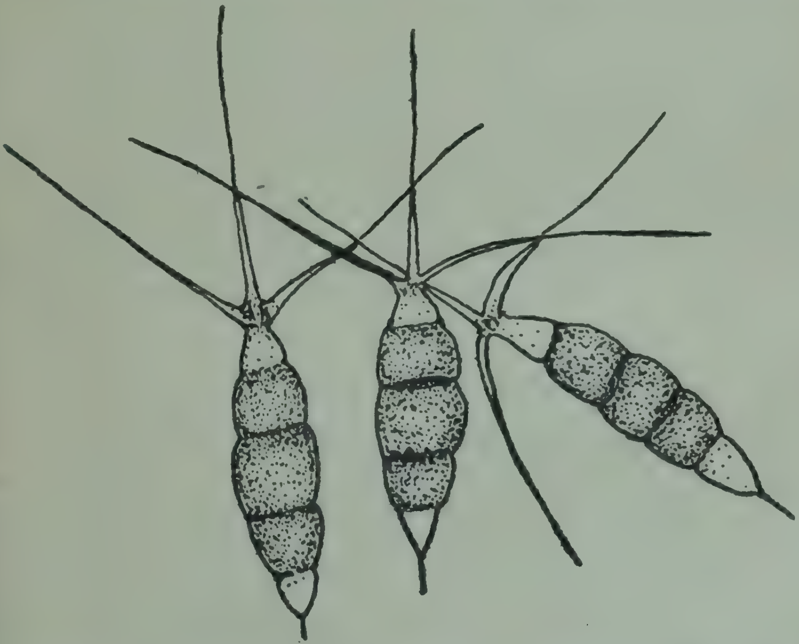
Geographic distribution. Bengal and Assam.

Appearance on the host plant. The spots appear as minute brownish areas on the upper surface of the leaves, especially the older leaves. Many of them coalesce and thus a considerable area of the leaves may be affected. As they become older they become grayish or whitish in appearance and become surrounded by brownish margins. Spots may also appear on the under surface but these rarely turn gray. Pycnidia occur mostly on the upper surface and along the margins of the patches. When infection is severe the leaves may fall.

The organism. The pycnidia are sub-epidermal, bowl shaped and measure 75-100 microns in diameter. Pycnospores are borne on stalks along the basal wall of the pycnidium. The stalk frequently comes away with the spore.

Pestalotia theae Sawada.

Pycnospores are 23-35×4·65 microns and the stalks are 3·1-9·3 microns. The pycnospores are slightly curved, fusiform, four septate and slightly constricted at the septa. The upper cell is crowned by three hyaline, filiform appendages which are slightly knobbed



Conidia of *Pestalotia* sp. drawn from a mount from a culture isolated from guava at the Agricultural Institute. Approx. 950 X.

at the ends. The appendages measure 6·2-40·3 microns. The three middle cells are dark coloured, (olivaceous) while the end cells are hyaline.

Control. Destruction of the infected leaves. Control measures are not well worked out for the disease.

CHAPTER XIV

SOME DISEASES OF COMMON MISCELLANEOUS CROPS IN NORTHERN INDIA

A large number of crops are grown in India which are minor in importance but which have their disease troubles just as the major crops do. A number of them will be given in the following pages.

Diseases of Sweet Potatoes

Although sweet potatoes are a common crop in many parts of India there is very little about the diseases to be found in literature. It is probable that there is much information of common knowledge that has not been written down for it is hardly likely that the sweet potato escapes attacks of the common fungi in India more than in other parts of the world. Bertus refers to *Sclerotium rolfsii* as causing a damping off of sweet potato in Ceylon. *Cercospera batatae* was reported on sweet potato a number of years ago. Mundkur (514) reported *Rhizoctonia solani* on sweet potato in the Bombay Presidency for the first time in 1934.

Rhizoctonia Rot

Hosts. A wide range of hosts.

Geographic distribution. World wide.

Appearance on the host plant. The symptoms of *Rhizoctonia* rot are not clear cut in all cases. There may be stem cankers, root infections or on the potato. Apparently the latter is the more common. At Allahabad *Rhizoctonia* was not found on the cankered roots and stems as often as *Sclerotium rolfsii* or *Sclerotium* *bataticola* although they are apparently equally distributed over the area.

The organism. *Rhizoctonia solani* Kuhn.

See under Irish potato.

The presence of the sclerotia on the surface of the potato will aid in the identification as they resemble those on the Irish potato.

Control. The disease appears to be minor in importance and thus is not likely to be often encountered.

Sclerotial Blight of Sweet Potatoes

Hosts: Widely distributed among the plants.

Geographic distribution. World wide.

Appearance on the host plant. On the sweet potato it is more of a damping off than any other. It is common in some countries in the seed beds in which case the stems are decayed just at or below the soil. The plants bear a typical damping off appearance. Some of the lower leaves yellow and the whole plant appears stunted. If there is moisture in the soil it often happens that there is a thin mat of white mycelium and in time it will be possible to see small round sclerotial bodies about the size and colour of mustard seeds. The diseased plants will pull away from the potato readily. Where there are sufficient plants, the mycelium will grow over the soil and contact a neighbouring plant thus spreading in the beds. In this way the fungus may destroy the plants in a fairly large area. It is this character that makes it so much easier to distinguish the *Sclerotium* from the *Rhizoctonia.*

The organism. *Sclerotium rolfsii* Sacc.

Hyphae are somewhat coarse with the cells, according to Harter and Weimer (272) being 2-9 × 150-250 microns. The sclerotia are at first white and then become brown, more or less smooth and glossy. They are more like mustard seeds than any thing that they may be compared with.

Infection takes place as the young plants are emerging from the soil. It is the young plants that are more readily attacked than the older ones. In the seed bed the mycelium covers the seed potatoes and it is no doubt in this way that the damping off occurred that Bertus (57) referred to.

Spores have not been reported for *Sclerotium rolfsii* and thus it appears the sclerotia are the major factors in the carrying over of the disease.

Control. One thing that makes the problem of control of Sclerotium diseases easier is that they are subject to the attack of saprophytic organisms and do not last long in the presence of organic manures. Fresh organic manures in the soil will build up the bacterial and fungus flora so that sclerotia will not last long. This is also true of the other fungi that attack the sweet potato in the soil.

Cercospora Leaf Spot of Sweet Potato

Host: Appears to be confined to the sweet potato.

Geographic distribution. India, Phillipines, Japan, Brazil, United States and probably other countries.

Appearance on the host plant. The spots are dark brown with light-centered areas that vary from 4 to 8 mm. They are irregular in shape and more or less limited by the veins. Some times the spots are dark or nearly black and extend through the leaf to both sides. In Java the disease is severe in wet seasons and in certain sections the sweet potato is a dry season crop. From the regions reporting the disease it appears that it is more a sub-tropical disease than of the temperate zone.

The organism. Cercospora batatae Zimm.

The fungus is typical of the *Cercosporas.* The mycelium is intercellular and limited by the necrosis of the tissues. Sclerotial masses form in the tissue and from some of these the conidia and conidiophores are produced. Conidia are 60-100×3-4 microns with considerable variation under differing climatic conditions. Harter and Weimer (272) mention that the spores from Florida were 50-150×4-5 microns.

Control. Welles (926) stated that in the Philippines spraying once every two weeks with Bordeaux controlled the disease. It is very mild in the United Provinces and probably causes insufficient damage to warrant spraying against.

Charcoal Rot

This disease caused by *Sclerotium bataticola* is common in many other parts of the world but appears to be of minor importance in India. The fungus, which has been discussed under the Irish potato and cotton, will not be given detailed treatment here. It is primarily a storage disease and not serious in the field, although it may be isolated from diseased stems and roots. At this time it does not seem to be serious enough to warrant more discussion.

Fusarium of Sweet Potato

Fusarium rot of sweet potatoes has not been recorded as severe. *Fusarium ozysporum* which has been recorded as attacking sweet potatoes in other countries occurs here and under some circumstances probably attack the crop in this country as well. However, it probably occurs only as a minor disease.

Rhizopus Rot of Sweet Potato

Species of Rhizopus can cause severe rotting of the roots when harvested and stored under condition favourable for the fungi. The roots should be subjecte to high temperatures for a short time after harvestin that the outer skin may be thoroughly dried and th excess moisture removed. This toughens the skin an makes it difficult for the fungus to penetrate.

Physalospora on Sweet Potatoes

This is a minor disease of sweet potatoes probably caused by *Physalospora rhodina*. It has been shown by Eddins and Voorhees (206) that *Physalospora ziecola*, *P. rhodina* and *Diplodia tubericula* can cause similar symptoms on maize and can cause typical pycnidia on sweet potato, watermelon, cotton, papaya, orange grape fruit and mango.

Cercospora Leaf Spot of Beets

Hosts: All members of the beet family and may also be found on many weed hosts in other families (898).

Geographic distribution. World wide.

Appearance on the host plant. On the leaves and petioles of the garden beet it apears as small circular somewhat water soaked spots which later become darker in the center and the margins often become dark or even reddish in colour. Spots may coalesce and thus a considerable area become infected but usually the damage is done by many small areas which kill the tissue. Infections on the petioles are likely to be dark in colour. Badly infected petioles of young beets may break at the point of infection. On young seedlings badly infected a damping off may occur.

The organism. Cercospora beticola Sacc.

The conidiophores are erect, under ordinary conditions they are short, simple and more or less olive drab in colour. They measure 35-55×4-5 microns. Conidial scars may be seen on the conidiophores.

The conidia are filiform, elongated, hyaline, multiseptate and vary from 75-200×3·5-4 microns. The conidiophores and conidia both vary in length and number of septations according to the humidity and temperature.

Infection occurs by way of the stomata and a single conidium may produce a number of germ tubes.

Control. Resistant varieties appear the best solution. Spraying and dusting has not been satisfactory. Destruction of the old waste material is one method of controlling, or at least reducing the disease.

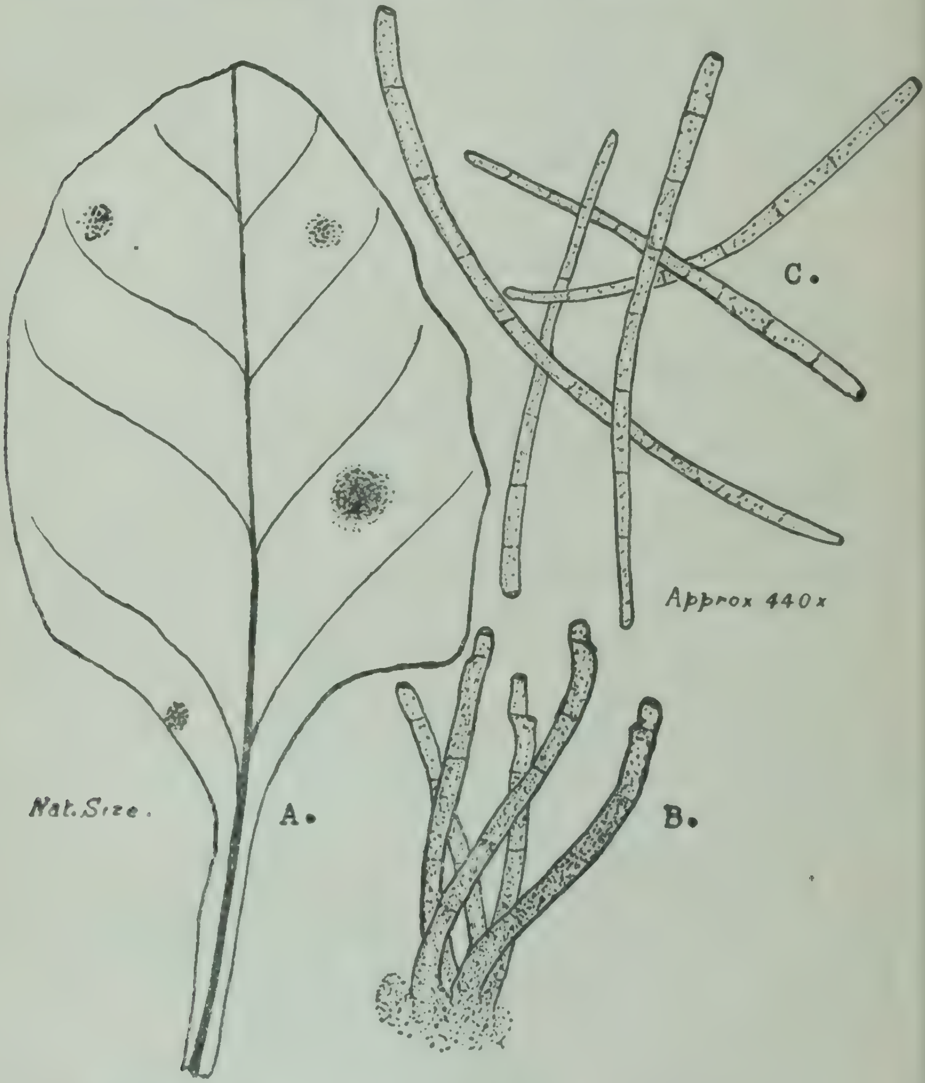


Diagram illustrating leaf spot of garden beet caused by *Cercospora beticola.*

A. Leaf with infection.
B. Conidiophores.
C. Conidia.

Phytophthora Leaf Spot of Colocasia

Host. It occurs on *Colocasia antiquorum* Schoot. It has also been reported on *Piper betle* by Thomson (811) and others, which would indicate it might be more common on that plant than generally supposed.

Geographic distribution. So far the fungus appears to be limited to this portion of Asia as no reports of its having been found widely disseminated have been made.

Appearance on the host plant. It appears on the leaf when the plant has attained a height of two to four feet. The height of the plant does not matter so much as the time of the year. At Allahabad, it usually makes its appearance about the end of the rains. At first the spots are small roundish and appear somewhat darker than the rest of the leaf. These spots increase in diameter rapidly and within a day or so are from a fraction of an inch to one or two inches in diameter. Several may coalesce and the whole leaf may be included in the diseased area.

As the spots increase they change colour slightly and small drops of a clear yellow liquid appear on the surface. A little later the center of the spots will appear brownish-yellow and there may be perforations in the tissue. The infected area soon dries and may fall off or break and become ragged. It is a common thing to find the margins of the spots zonated with various shades of green and brown and, although the disease represents a serious loss to the grower, it presents an interesting study of colours.

In mild cases the leaves may have only scattered spots, while in severe cases the leaves may be completely destroyed. During the past few seasons at Allahabad the leaves have been heavily spotted and in localized areas there has been serious damage to the crop. If the petioles become infected the leaves may break and fall and this hastens the damage. Complete destruction of leaves and petioles has been observed where the disease becomes epidemic.

The corm may develop a rot in storage which resembles that of late blight of potatoes. It is probable the infections result from the spores which fall on to the ground or are shaken on to the corms at harvest time from the infected leaves.

The organism. Phytophthora colocasiae Rac. Leonian (375) placed it in the species *Phytophthora parasitica* Dastur; but Ashby, (34) reviewing the work, concludes that there is no justification for placing it under the name *P. parasitica* and that it should be retained as *P. colocasiae*, a distinct species.

Life cycle. The fungus is intracellular, possessing large hyphae which are much branched within the host tissues. The hyphae feed by means of haustoria which penetrate the cells and absorb the food material. The hyphae have not been observed to enter the vascular bundles but appear to be confined to the softer parenchyma tissues.

The sporangia are borne on short stalks that emerge from the stomata. These are unbranched and are usually about 50 microns in length and not more than 1 to 2 microns in diameter .The sporangiophores may emerge singly or in clusters. The conidia (sporangia) are colourless and somewhat pear shaped, with the blunt end attached to the conidiophore. They measure from 38-60×18-26 microns. They are thin walled and at the free end there is a point which expands in water and permits the zoospores to escape. There are upwards of 20 zoospores in a single sporangium. From observations it has been found that the higher temperatures restrict the zoospores formation and that below 80 degrees F. it occurs freely. If for any reason the sporangia do not come in contact with water immediately they may germinate directly by means of a germ tube. This tube is capable of infecting the Colocasia plant and thus there is double source of infection.

Oospores and antheridia are formed in the tissues of the host plant. The oogonium is nearly round and usually somewhat yellowish when mature. It measures from 24-35 microns in diameter. The oospore is from 4-8 microns smaller. It is certain that the oospores are the resting spores of the fungus and that they are a factor in passing the disease from one crop to the next.

The fungus may be initiated by the oospores or by the mycelium in the corms. The attack once begun in the leaves spreads rapidly and from the time of the entrance of the mycelium until the appearance of the conidia on the surface may be only three days. As the disease is spread by means of the conidia during the growing season, it is readily seen that, under favourable conditions, an epidemic may spread so rapidly over the field that it would appear to have sprung up all at once.

Control. Su (752) reports that in Burma the leaf spot was controlled on *Piper betle* by the use of 1% Bordeaux. But it has not been generally used for the leaf spot of *Colocasia.* Clean seed and rotation of crops coupled with sanitation are probably sufficient for most of the seasons. Since the fungus is also found as a cause of foot-rot of *Piper betle* it will be referred to under that heading also.

Bacterial Soft Rot of Poppy

Host. Appears to be confined to the poppy.

Geographic distribution. So far the only report found is the one from Pusa by Ram Ayyar (635) although there is another bacterial disease reported from the United States which was named *Bacterium papavericola.* The differences between them are too great, however, for them to be confused.

Appearance on the host plant. The stems become rotted, blackened, then followed by internal disintegration. In the advanced stages the plant exudes a slimy mass of bacteria.

The organism. The organism was named *Bacterium papaveris* by Ram Ayyar (635). He describes it as a short rod, actively motile by four to eight peritrichous flagella; Gram-negative; coagulates milk; forms acid in peptone and with optimum temperature of 30°C.

Control. Destruction of diseased plants and rotation of crops. Selection of resistant varieties and control of the insect agent of transmission (*Scirpophaga*).

The Downy Mildew of Opium Poppy

Host. The disease has been found on the opium poppy (*Papaver somniferum* L.) and the Mexican poppy (*Argemone mexicana* L.) as well as a number of other members of the genus *Papaver.*

Geographic distribution. It appears widespread. It is found wherever the host plants are grown, which is generally throughout the world within the tropical and sub-tropical regions.

Appearance on the host plant. About the beginning of February—although in some cases the fungus has been reported as early as November—pale brown spots appear on the leaves of the wild and garden poppy, chiefly along the margin or near the tip. If conditions are favourable they may spread over the whole leaf. The leaf will then dry up and become brittle and easily broken. In the vegetative stage the conidiophores may be so dense that together with the conidia they give a grayish-violet colour to the spots.

The organism. Peronospora arborescens (Berk.) de Bary.

It appears that this species also has biologic strains as Yassifovitch (940) concluded that *P. effusa* var. *papaveris*, *P. grisea*, and *P. papaveris* are synonyms of *P. arborescens.* He also believes that *P. arborescens*, which he found in Yugoslavia, is also a biologic form as it does not attack the cultivated poppy.

The mycelium is typical of the other members of the *Peronospora.* Conidiophores are in groups of five or six. They are somewhat longer than most of the conidiophores and are sturdy and erect, sometimes as much as 1 mm. in length and 10-12 microns wide. They branch dichotomously and may have ten to twelve branches. The top branches are very fine and slightly recurved. The conidia of the poppy mildew are more nearly round than the other conidia of the *Peronospora.* They are from 20-25×18-22 microns and of a pale violet colour. Germination is direct by either a terminal or a lateral germ tube.

Oospores are formed in the dead and browned tissue. These are some 33 microns thick and consist of a thin oogonial wall within which is a layer of reddish-brown periplasm which varies rom 4-5 microns in thickness. The oospore averages some 26 microns in diameter.

Life cycle. The life cycle is typical of the other members of the genera *Peronospora.* Oospores germinate by a germ tube in the beginning of the vegetative season and infection takes place at the time of emergence from the soil of the little plants. There is also the presence of wild host plants which are infected and from which conidia may be blown to the field crop. Scattered by wind and rain the conidia spread the disease during the growing season. As the leaves die the oogonia and antheridia are formed with the oospores following immediately.

Control. Sanitation and clean cultivation are the only remedies generally practicable. Resistant varieties are a possibility.

Stem Rot of Jute

Hosts. The organism has a wide range of host plants, including, in addition to Jute mentioned above, potato, cotton, pigeon pea, ground nut, alfalfa, tobacco, mung bean, mulberry, sesamum, brinjal, cowpea and orange.

Geographic distribution. It has been reported in nearly every country of the world and from many parts of India.

Appearance on the host plant. Varada Rajan and Patel (867) have made a more or less complete report of the organism on jute in India. On the seedling plants the disease manifests itself as lesions on the collar or on the cotyledons. The lesions are dark in colour and are in the shape of thin streaks. The disease may become so severe in damp weather as to cause the seedlings to damp off. Under dryer conditions the symptoms are a wilting or a blight.

The first infections usually appear on the above ground parts but the roots may become infected later. Leaf infection may be followed by the formation of pycnidia (*Macrophomina stage*) with the sclerotia forming later.

The lesions, which are more or less buff in colour, form along the margins and at the apex of the leaves. The entire leaf may be covered with lesions. Even the midrib and the petiole may be included. On the stems the lesions are more likely to occur at the nodes. These are small at first, later increasing until the stem may be girdled, in which case the plant wilts, the leaves fall and only the bare stalk remains. In elongated lesions the tissues may shred and thus assume a ragged appearance. Occasionally adventitious roots may form at the edge of the cankers, but the plants almost never really recover but remain dwarfed and stunted.

If the roots are infected they rot and are found filled with sclerotia.

Where infection extends to the inflorescence the capsules are discoloured and the seeds are diseased. Such seeds are lighter in weight than normal and they have a dirty, dull appearance. Sclerotia may be found on the seeds or capsule.

The Organism. Macrophomina phaseoli (Maubl.) Ashby or *Rhizoctonia bataticola* (Taub.) Butler. The term *Sclerotium bataticola* Taub. will also be encountered in literature as it has been observed (Hansen and Valleau (281) that the pycnidia formed are *Ascomycetc* like. Pycnospores measure 15.6 to 27.3 microns. The sclerotia vary from 41 to 86 microns.

Control. Rotation of crops. This is not so easy with the large host range of the fungus. Deep plowing to bury the sclerotia would help.

Leaf Spot of Eleusine Coracana

Geographic distribution. Common throughout India on species of *Eleusine.*

Appearance on the host plant. The disease may appear on the root, stem and all parts of the leaf and may also cause death of the seedlings. The disease shows on both sides of the leaves as small oval, mineral brown spots which gradually elongate parallel to the veins. They later become dark chocolate brown. Mature spots are 10-15 mm. in diameter, but may coalesce into large irregular patches. Leaf shredding does not occur as in the case of some other leaf spotting species of *Helminthosporium.*

Spots on the leaf sheath are not well defined as on the leaves. They are usually larger and of a chocolate brown colour. They will usually be found at the junction of the leaf sheath and blade. Spots on the stem are of an oval shape and more or less oblong. Sepia in colour. The cortical tissue is invaded and in time may collapse, resulting in the death of the tissue above it. Severely diseased spikes are sterile. Healthy spikes will be observed to be recurved at maturity and the diseased ones will remain errect. In moist weather the diseased spikes are sooty or olivaceous in colour.

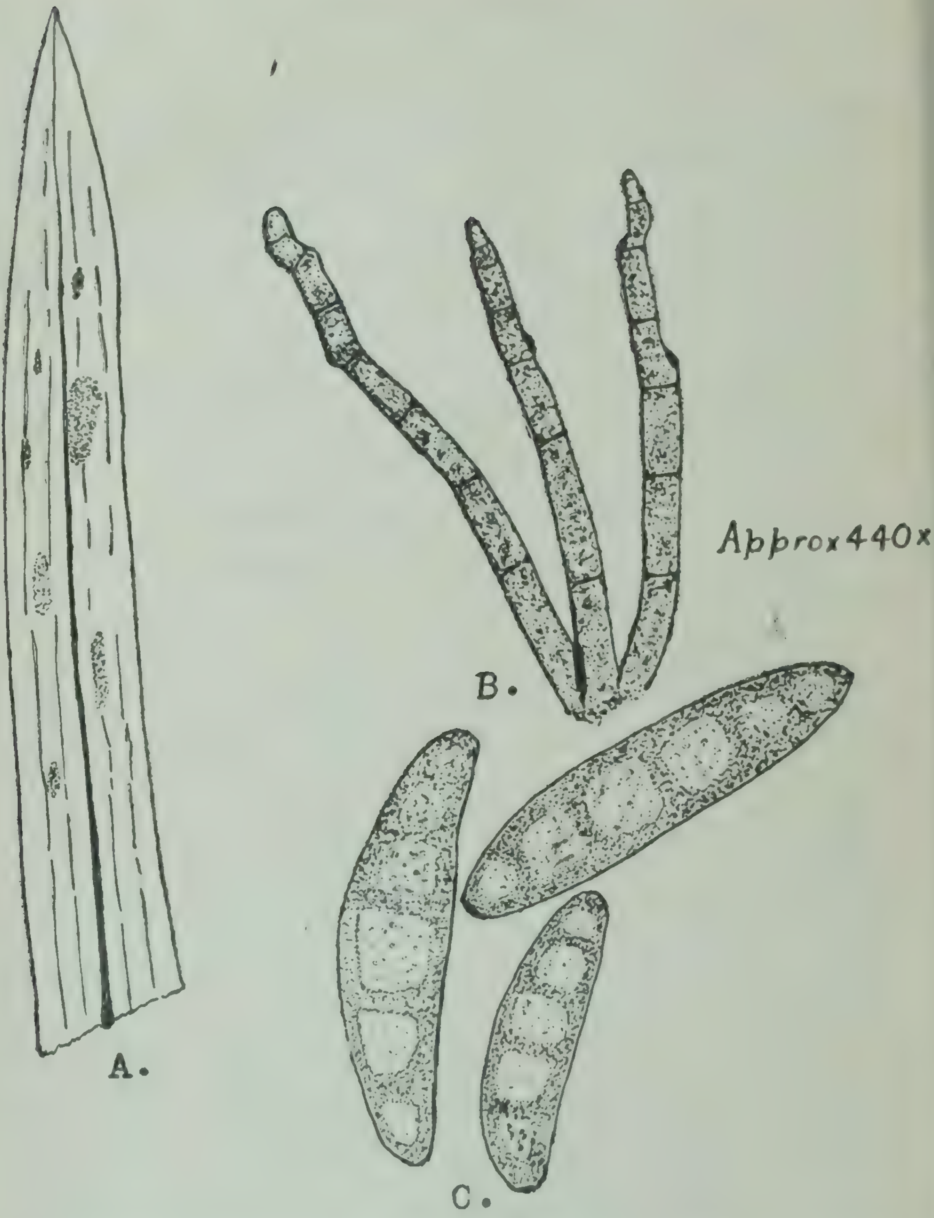


Diagram illustrating leaf spot of Mandua or Ragi (Eleusine coracana).

A. Infected leaf.

B. Conidiophores.

C. Conidia.

The organism. Helminthosporium nodulsum. Mitra and Mehta (491) have given a very complete description of the organism. The mycelium is septate, sub-hyaline being both intra and inter-cellular. There are no haustoria. The hyphae average some 4 microns in diameter. The conidiophores are tout, erect and rigid, arising singly or in clusters of two to seven, which emerge from the stomata, more rarely through the epidermis. They may arise from creeping hyphae on the surface of the host. They are bulbous at the base, mostly unbranched, often curved and with prominent geniculations. In colour they are dark brown to brown at the base with a hyaline to sub-hyaline tip. Septa 4-18, average 9. They measure 12-356×3·5-8 microns with the average 150·6×6 microns.

The conidia are thick walled, subhyaline. They are sub-cylindrical to ovate, rarely subclavate, slightly curved or straight, widest near the middle. They taper towards both ends which are rounded off abruptly and with a conspicuous hilum. Mature spores are light russett green with the end cells somewhat lighter in colour. They measure 40-114×11-12 microns with 3-11 septa. The average size being 67×14 microns with 8 septa.

Smut of Ragi

Host plants. Reported on ragi (*Eleusine coracana* Gaertn)

Geographic distribution. Mundkur and Thirumalachar (530) state that the disease has been recorded in the ragi growing areas of Mysore, Bombay and Madras Provinces as well as Kenya and Queensland.

Appearance on the host plant. The disease appears as enlarged grains scattered over the spikelets. These are five to six times the normal size and slightly greenish in the early stages becoming darker as they become older. The sori within the enlarged seed mass are inclosed with a thin membrane which, according to the above mentioned authors, gradually turns pinkish-green and begins to rupture.

The organism. Melanopsichium eleusinis (Kulkarni) Mundkur and Thirumalachar (433). According to the same authors (Phytopath. XXXVII, pp. 481-486, 1947) the smut had been named *Ustilago eleusinis* by Kulkarni but a comparison of the smut of ragi with that of the gall forming smut of *Polygonum glabrum* (*Melanopsichium pennsylvancium* Hirsch(convinced them that this organism could not be *Ustilago*, hence the above name.

The spores are produced in a mucilaginous matrix which is set free. Chlamydospores are globose to sub-globose, measuring 7-11 microns in diameter with a pitted epispore. The chlamydospores germinate to produce a septate promycelium with both primary and secondary sporidia.

Control. Air borne spores appear to be the method of dispersal. Seed treatment is not effective. Rogueing of the smutted ears is effective though slow and tedious. Deep plowing has been suggested as one means of reducing the infection.

Paspalum Smut

Host plants. On species of *Paspalum*, especially *P. sacrobiculatum*.

Geographic distribution. The disease has been recorded in Ceylon, Australia and India.

Appearance on the host plant. The first symptoms of this disease are in the ear. The whole structure is transformed into a mass of fungus tissue which is, at first, surrounded by a delicate cream-coloured membrane. Only the fibrovascular bundles remain of the inflorescence.

The organism. Sorosporium paspali McAl.

This smut is similar to the other species of the genus *Sorosporium* in the formation of the spore balls which, according to Butler (93), Measure from 30 to 50 microns in diameter. The spores are roughly pear shaped and measure from 11 to 18 by 8 to 12 microns. McRae (444) reports that some 2% of the spores will germinate in the first 4 months and that 90% of them will germinate about 9 months.

Control. McRae (444) state that a 1% copper sulphate solution inhibited the germination of the spores but that it required a 5% solution to kill them. He was able to reduce the amount of infection by 50% with 1% copper sulphate dust.

Wilt Disease of Safflower

Safflower (*Carthamus tinctorius* L.) is a common plant over much of India. It is not grown in large acreages in one area but is used as a border crop or to separate two fields or sections of the same field. It is a border crop because some varieties, are spiny and the spiny leaves discourage the neighbouring cattle. The most serious disease of safflower in many sections is the wilt.

Hosts. The fungus (*Sclerotinia sclerotiorum*) is wide-spread over the world and has a wide host range.

Geographic distribution. World wide.

Appearance on the host plant. The first symptoms are a white yellowing of the leaf tips which may spread rapidly and soon result in death of the whole plant. If the collar is examined it will be usual to find a dense growth of mycelium which is whitish in colour. Later there will be numerous black sclerotia which range from 2 to 12 mm. formed over the roots and more or less loosely attached to them. If the stem is cut open similar sclerotia will be found there. Joshi (325) described the disease in 1920 and mentions that the pithy portion of the inner stem will shred easily when diseased. In fact the entire portion of the stem shreds.

The fungus is systemic and often there are sclerotia formed in the base of the flower heads and these will break easily.

The organism. *Sclerotinia sclerotiorum* (Lib.) de Bary.

Mundkur (509) in 1934 described the fungus on *Hibiscus* at which time he was able to secure the perfect stage by placing the sclerotia in a refrigerator for several weeks. He states the apothecia are at first fawn coloured, saucer shaped, later becoming flat or convex. They become darker with age and finally become a chestnut brown. The stipes were from 20 to 45 mm. long.

The mycelium is abundant and of a whitish colour.

Control. Destruction of the diseased plants so that the sclerotia do not remain in the soil. Careful rotation. Some of the cereals appear to be immune as it has not been reported on jowar, bajra or maize. It has been recorded on wheat however and so it may be that the fungus is so wide in its host range that rotation is almost impossible as a control measure. As it is a root disease, (at least on the below ground parts) it is probable that organic manures and resistant varieties hold the best hope of control where it is a factor in production.

The Downy Mildew of the Soy Bean

Host. In the Ganges Valley it has been found on the wild species of *Medicago* and *Meliotus*. It has been reported on *Glycine hispida*, *Medicago denticulata*, *M. lupulina*, *M. alba* and *M. indica* by Butler (93) in India. Lehman and Wolf (373) find a downy mildew on the soy bean in America which they consider different from the one here. Wolf and Lehman (936) name it *P. sojae*. Butler and Bisby (96) think that the one in India is much more nearly *P. trifoliorum*.

Geographical distribution. If the closely related forms are considered, it appears to have a very wide range of host plants and a wide geographical distribution.

Appearance on the host plant. In general appearance it is very similar to the downy mildew on the field and garden pea. The same chlorotic type of spots appear on the upper surface and the gray violet cottony growth on the lower surface. The affected leaves wither and then fall before their time.

The organism. Peronospora trifoliorum de Bary. The same general characters are found in the fungus causing the downy mildew of soy bean as for that causing the mildew on the field and garden peas. Conidiophores are from 300-450 microns in length by 9-11 microns broad. The branching is very much the same, being from 6-8 times. The tips of branches are less curved than those of *P. parasitica,* and the terminal branches diverge at nearly right angles, are pointed and the conidia are borne at the tip. These are more nearly like those of the poppy mildew than *P. viciae,* being nearly round, or broadly elliptic, 18-24×15-18 microns and of the same pale violet colour as *P. arborescens.* They measure rom 24-31 microns and are enclosed within a thick wall which is light brown in colour and smooth.

Mahali or Koleraga Diseases of Areca Nuts and Coconuts

As the two diseases, or, perhaps, it is better to say the same disease, can be found on both plants the symptoms as observed on the two will be discussed at the same time.

Host. Areca catechu, the betle nut, the coconut, *Cocos nucifera,* and the palmyra palm, *Bosarrus flabellifer.* Other hosts may exist but these are the commonly recorded ones.

Geographic distribution. The areca nut and coconut palms are grown mostly in the South India and West India areas which include Mysore, Cochin, Travancore and portions of the Kanara and Malabar districts. The disease is prevalent in all of these areas.

Appearance on the host plant. The most typical symptom observed on the *Areca* nut is the dropping of the immature nuts. Examination will show diseased areas over the nuts which are covered with a white cottony growth. All of the nut or only portions may be diseased. Infections are commonly found on the stem end of the nuts. The fungus may also penetrate into the crown of the palm by way of the pedicels and in this way the whole crown may become diseased.

On the coconut the young leaves and shoots are attacked. Ashby (34) considers that there are two conditions that exist in the diseased plants. In one case the central bud rots from the attack of *Phytophthora palmivora* and in the other case rotting is due to a bacterium. He believes that the species of *Phytophthora* which he found in the West Indies is the same as the one found in India.

The organism. Phytophthora faberi Maubl., *P. meadii* MsRae, *P. palmivora* Butler. The above three so-called species have been reported as causing the disease of areca nut and coconut palms. Lester-Smith (376) after cultural studies considered that *P. faberi* and *P. palmivora* are the same and should be called *P. palmivora* because of priority. *P. arecae* (Coleman) Pethybridge has since been changed to *P. palmivora* by Leonian (374). It was reported as one of the causes of the coconut diseases by McRae (437). In addition to the above, Verghese (897) states that in Travancore the leaf stalk rot of coconut is caused by *P. parasitica*, the bud rot by *P. palmivora* and the fruit rot by *P. arecae*. Ashby (34) after studying the paired cultures of *P. arecae* and *P. meadii* believes they are the same but that they should be called *P. arecae* because of priority.

The question of nomenclature is a perplexing one and it will be seen from the above that as yet no agreement has been reached by the various workers. Some new light was thrown on the subject by Narasimhan (538) who studied isolations from various plants alone and paired with similar and dissimilar strains. He collected 7 strains of *Phytophthora* from plants in Mysore as follows: *Santalum album* L., *Loranthus longiflorus* Desv., *Jatropha curcas* L., *Bryophyllum calycinum* Salisb., *Artocarpus integrifolia* L., *Colocasia antiquorum* Schott. and *Ficus hispida* L. He had *Phytophthora arecae* in culture. When the areca or Loranthus strains were grown together with one from *Santalum* or *Jatropha*, oospores were formed at the juncture of the cultures. When *Santalum* and *Jatropha* strains were grown together, or when areca and *Loranthus* strains were grown together no oospores were formed. He considers this to be an indication of heterothallism. Cultures *P. parasitica* and *P. meadii* that had lost the power to produce oospores, when grown with a male strain (in this case *P. arecae*) formed oospores but did not when grown with the strain from Santalum. Those forms which did not produce oospores when paired with themselves were assumed to have lost the other sex. In support of this theory is the work of Uppel and Desai (856). They found that strains of *P. arecae*, causing the kolerage disease in North Kanara, Bombay Provinces, did not form oospores by themselves but did when paired with strains from different localities. They concluded that the sexual strains had become separated and thus oospores were formed only when they were brought back together. This fact made the explanation of the perpetuation of the disease in the various districts difficult. The work of Galloway (238) is of interest in this respect. He used strains of *P. meadii* and *P. colocasiae*, which had formed oospores, but which had lost the power, growing them upon filtrate of strains which formed oospores. *P. meadii* formed oospores at 23°C. *P. colocasiae* formed a few at higher temperatures, but only a few. But this experiment does throw some doubt on the theory of heterothallism.

Conidial, oogonial and oospore measurements are nearest those of *Phytophthora palmivora* than of other species of *Phytophthora.*

Life cycle. The fungus which causes the disease of areca and coconut palms appears to be carried over the dry seasons in the old dry diseased fruits if oospores are formed. If they are not formed, as in the case of some of the sections where it is held that only male or female strains are present, it is difficult to explain the perpetuation of the disease. Here it may be in the mycelium stage in the margins of old diseased areas.

After the first infections have started in the beginning of the rainy season, the production of sporangia and zoospores will carry the disease from tree to tree and to new spots on the same tree. McRae (437) shows that tappers other than man, the rhinoceros beetle (*Orycles rhinocecros* Linn.), and rain are also agents in spreading the disease. He was able to show that the bud rot of the coconut palm (*Phytophthora palmivora*) can also infect the Palmyra palm and it does go back and forth between the two. Although oospores are not commonly found, yet they are found in sufficient numbers and times to make them a factor in the spread of the disease from season to season.

Control. McRae (437) gives the history of the operations against bud-rot of palms in South India up to 1923. Early control was largely confined to cutting the diseased palms and burning them. This began in 1906. From the first cutting of the diseased palms until 1921, 956,446 were cut down and burned. At the same time, 136,693 had the outer leaves removed and these burned. From 1913 until 1921, 99,938 trees with internal infections were treated by cutting out the diseased portions and burning them. During that period over 2,800,000 palm trees were examined. In 1920 the Pest Act was passed and this legislation placed the responsibility of the examination and destruction of the diseased trees upon the owner. As a result the Government examinations in 1920 were only 16,000 and in 1921 only 8,000 trees.

By this time the spraying of the palms with Bordeaux had become quite general and with the improvement of the spray mixtures and introduction of new methods became more and more effective. In 1922 Narasimhan (Journ. Mysore Agric. and Exper. Union, V, p. 1-4, 1922) gave a modified formula for Bordeaux which contained the regular 5-5-50+2 pounds resin +1 pound of soda. The resin and soda are dissolved in hot water and added to the Bordeaux. Casein was also added as a spreader for the Bordeaux. Venkata (887) reported that lime-casinate added to the Bordeaux was very effective. Narasimhan (537) reported a new spray which proved very effective against the kolerage disease. This spray was known as Martini's Bordeaux. The formula is as follows:

(A) 2 ℔s. potash alum
2 ℔s. copper sulfate in 12 gal. water
2½ ℔s lime in 12 gal. water.
The lime solution and the potash alum-copper sulphate solutions were added together.

(B) ½ ℔. casein in ½ gal. water.
½ ℔ lime in ½ gal. water.

These were mixed together and then added to solution (A) resulting from the potash alum-copper sulphate mixture above.

He reported that this solution was effective and cost at that time only about Rs. 2|- per acre. Narasimhan reported in 1931 that the addition of 2 fluid ounces of castor oil per pound of solid in the spray gave excellent results.

In the first attempts to spray for a number of years afterwards the spray was carried to the tops of the palms on the backs of the labourers. This was slow and meant often that the work was poorly done because of the low pressure in the spray tank. In 1928, Narasimhan (537) reported that by allowing the labourer who climbed the tree to carry a long hose with him and keep the spray outfit on the ground a much better pressure and more spray material could be supplied. Whereas by the old method only about 300 trees could be sprayed, by the new method some 1,500 trees could be sprayed. The same author also reported the use of peanut oil in place of castor oil with excellent results.

The brief review of the control methods will give some idea of what is being reommended at this time and the methods that appear most effective.

Pod Rot of Rubber Trees

Hosts. Recently the work of a number of men has led to the belief that the species of *Phytophthora* which caused the pod spot and canker may have a wide range of host plants. In addition to species of *Hevea* it is also thought that the coconut palm, cacao and the palmyra palm are also victims of the *Phytophthora* which cause the pod and bud rot.

Geographic distribution. The disease is widely distributed but it may be that the same disease may be caused by different fungi in different parts of the world. In India, *Hevae brasiliensis* (the rubber plant) is grown principally in the southern portion of the country. In 1918 McRae discussed the disease as caused by *Phytophthora meadii.* Since then it has been reported from the rubber growing areas of the tropical zone.

Appearance on the host plant. There are a number of distinct symptoms of the disease on the rubber trees. Wilting of the leaves, fruit-rot, leaf-fall, bark-rot and die-back are recognized as evidence of the presence of the fungi. These symptoms begin to appear about the middle of June after the monsoon has broken. At that time the fruits begin to show dull gray ashy spots on the surface. This spotting may begin at the pedicel end of the fruit and extend downwards until the whole fruit is involved. There may be several small spots which run together, with drops of ooze appearing on the surface. These turn black with age. Soft rots follow and the fruit becomes soft and soggy, later splitting along the suture and exposing the hard endocarp within, which does not split. The fruits may fall soon or may hang on the trees for some time, even after the season is over. After the rains have stopped for a few days the surface of the infected pods becomes covered with a thin layer of downy growth which is a dense growth of conidiophores and conidia of the *Phytophthora*. In epidemics every tree may be infected and every fruit on a tree.

The disease on the bark is known as canker and is less conspicuous than the fruit rot. On young trees the bark may appear darker than normal, but this may not be seen if the stem is old enough to take on the dark brown normal colour. In some cases there is a reddish exudate which appears in damp weather and aids in locating the spots. However, as this will likely happen only on large areas, the small areas are left undetected. As no latex is produced in the diseased area the first indication that there is disease may be the stopping of flow in tappings below the canker. If the bark is scraped away the under tissues will be found discoloured. The laticiferous tissue is discoloured and beneath there is a grayish colour with a distinct black border. The latex in the tubes is coagulated and if a canker is cut will be a dirty red at first and later become darker. When the current leaves have fallen the young branches die back, which causes a secondary growth. The more rapid the growth, the more rapid the die-back. This die-back is one of the symptoms of the disease.

As new leaves and shoots appear there is a secondary invasion of the fungus and the secondary leaf-fall takes place. This is serious as it causes the loss of stored food for the succeeding season's growth.

Staughton-Harris, who worked in Ceylon for the rubber industry, found that the conditions in South India were much more severe than in Ceylon and that as a result there was much more of the disease there than in sections to the north. This secondary leaf-fall usually begins to appear on the trees about the 5th year after planting. Occasionally it appears as early as the 2nd year, but that is rare.

The bark-rot is much more likely to be connected with the tapping operations than with natural infections. It has been shown that deep tappings produce more of the bark infections than shallow tappings.

The organism. At least two species (so called) have been associated with the diseases of the rubber tree. Pod-rot and canker have been thought to be caused by *Phytophthora faberi* Maubl, while the black thread disease has been said to be caused by *Phytophthora meadii* McRae. This, however, depends upon the acceptance of the validity of species of fungi (*Phytophthora*) now in literature. Tucker (823) combined *Phytophthora aberi* and *P. meadii* with *P. palvimora.* If this is accepted, then there is only one species responsible for the diseases of rubber. It will be seen from the following discussion of the diseases of the areca nut and coco palm that the same species of *Phytophthora* may also be responsible for some of the diseases on them. Biologic strains are hinted at. Narasimhan (538) suggests heterothallism as a possibility and thus an explanation of the appearance of similar organisms on different host plants. Uppal and Desai (856) also suggest this as a possibility. For the present time it seems that no one of the names may be used with positive assurance and that any reference to the diseases would be safer if only *Phytophthora spp.* are given as the causal organisms.

The mycelium is hyaline and variable in diameter, usually from 2·5-6 microns. The hyphae are at first intra-cellular and later inter-cellular. Haustoria are rarely found. The sporangia are terminal but as growth of the sporangiophore continues it is left to one side, as in the case of *Phytophthora infestans,* but without the swelling beneath the point of insertion. The sporangia are egg-shaped or lemon-shaped, hyaline, with a prominent papilla at the tip and are 30-60 × 21-30 microns. Occasionally they reach 80 × 42 microns in size. They germinate to produce zoospores and may produce as many as 30. In some cases secondary sporangia are formed on germ tubes from the primary sporangium. Chlamydospores have been found as in *P. colocasia.* These are spores that are vegetative in character and may be formed anywhere in the mycelium by a swelling of the hypha and cutting off of the swollen section by cross walls on either side. This portion then rounds up and a wall is laid down around the contents, making a spore. These behave as ordinary spores and germinate under favourable conditions to produce additional mycelium. These have not been observed outside of cultures.

Oospores are formed in the old infected portions of the plant. They were first found in old rotted fruits under trees. Only a few are found and it appears that they are not formed in large numbers in nature. This may be explained on the basis of heterothallic forms as referred to above in this discussion. The average size of the oospores appears to be 25·5 × 24·9 microns. The oogonial wall is very thin and there is little difference between the size of the oospore and the oogonium.

Control. The best control so far has been secured by the use of Bordeaux. Ashplant (35) states that the use of Bordeaux will control the disease known as secondary leaf-fall in South India on *Hevea* at a cost of from Rs. 8 to 17 per acre.

Butler (93) refers to a "black thread" disease of rubber which has been serious in Burma. According to Butler this disease is caused by *Phytophthora meadii* McRae. Leonian (375) has merged this organism with *Phytophthora palmivora.* His work, however, has been criticized by others and perhaps should not be taken too seriously. Tucker (823) does agree with Leonian in the placing of *P. faberi* and *P. meadii* with *P. palmivora. P. meadii* is also associated with the disease of coconut palms in South India as well as the areca nut disease. Recent literature does not stress the "black thread" disease and it would appear that it might be a form of the disease which affects the leaves, fruits and young branches. This would be dependent upon the acceptance of the causal organism's mergence with *P. faberi* into *P. palmivora.*

Butler recommends some disinfectant being applied to the cut surface when tapping and increasing the distance between trees in the planting. Bordeaux paste or spray would be effective against the fungus as it has been effective against the same organism in other cases, namely on Cacao.

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PERIODICALS WHICH MAY CONTAIN INFORMATION OF VALUE TO THE STUDENT

Just as the student may be at loss for a good reference book, so will there also come times when periodicals will be needed for the current information along many lines of plant pathology and mycology. The following list is a selection from the many that have been, or are being, printed over the world. The Indian publications are listed first and the foreign afterwards.

INDIA :

Agriculture and Livestock in India (Now Indian Farming)
Allahabad Farmer and other college magazines.
Current Science.
Indian Journal Agricultural Science.
Indian Farming (formerly Agriculture and Livestock in India)
Indian Phytopathology.
Journal Indian Botanical Society.
Madras Agricultural Journal.
Memoirs Department of Agriculture, India Bot. Series.
Publications of the various Universities such as Punjab, Bombay, Calcutta, Allahabad and others.
Proclamations of the Indian Academy of Science.
Provincial publications such as the bulletins published by the United Provinces Department of Agriculture.
Reports of the Indian Agricultural Research Institute, the Director of Agriculture of Madras, Ceylon, Bombay, Assam, United Provinces, Orissa etc.
Science and Culture.

GREAT BRITAIN :

Annals of Applied Biology.
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CANADA :

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FRANCE :

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ITALY :

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IRELAND :

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GERMANY :

Annals of Mycologici.
Centralblatt fur Bakteriologie.
Deutsche Zukerindustrie.
Phytopathologische Zeitschrift.
Zeitschrift fur Pflanzenkrankheiten.

RUSSIA :

Bulletin Plant Protection.

UNITED STATES :

American Journal Botany.
American Potato Journal.
Botanical Review.
Botanical Gazette.
Journal Agricultural Research.
Journal American Society Agronomy.
Journal of Heredity.
Mycologia.
Plant Disease Reporter.
Phytopathology.
Science, N. S.
State Experiment Station publications.
United States Department of Agriculture Year Books.
United States Department of Agriculture Bulletins and Circulars.
University and College publications.
Various other publications such as the State academy of science reports, Boyce-Thompson Institute, etc.

AUSTRALIA :

Journal Agriculture, Victoria.

Journal Ministry Agriculture, West Australia.
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MISCELLANEOUS PUBLICATIONS :

Agricultural Gazette, New South Wales.
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Journal Department Agriculture, South Africa.
Lingnan Science Journal, China.
Malayan Agricultural Journal.
Nanking Journal, China.
New Zealand Journal Agriculture.
Philippine Journal Science.
Porto Rico Journal Agriculture.
Tropical Agriculture, Trinidad.

GLOSSARY

Acervulus. A saucer-shaped structure composed of mycelial threads which may bear spores at the tips. The typical asexual fruiting structure of the Melanconiales.

Acicular. Slender. Needle shaped.

Acrogenous. Growing at the apex.

Acropetal. Producing at the apex in a succession. Resulting in a chain.

Adnate. Attached along the entire length.

Aeciospore (aecidiospore). A spore formed in an aecium. In the black stem rust wheat the spore resulting from the union of sperm and egg. The aecidium is the direct result of the union and the spores are the secondary result.

Aecium (accidum). The cup formed in the barberry leaf as a result of the union of sperm and egg.

Aerial. Growing in the open air. Living on the surface of some material so the branching parts and fruiting structures are mostly in the air.

Aerobic. Organisms requiring free oxygen to live.

Aethalium. A structure which is composed of many plasmodia. Being compound.

Agglutinated. To glue together. In the case of bacteria they are caused to clump as a result of the action of certain substances bearing electric charges and known as agglutins.

Aggregate (aggregated). To collect or mass together.

Allantoid. Shaped like a crescent but with rounded ends.

Alveolar (alveola, alveolae). Cavities or pits in the body surface.

Alveolate. Being honeycombed with pits.

Amoeboid. Exhibiting a creeping movement like an amoeba.

Amorphous. Without shape, powdery.

Anastomosing. Forming a network by fusing together.

Annulate. Ring-like or with a ring-shaped structure.

Annulus. A ring-like portion of the ruptured marginal veil covering the pileus of the mushrooms before they expand.

Antheridium. The male reproductive body of some of the fungi, algae, mosses, liverworts and ferns.

Antigen. A substance that can cause the formation and appearance of specific antibodies. An antibody is a body that will neutralize a foreign substance (usually protein in nature) when it is introduced into the animal blood stream.

Antitoxin. A secretion of the living protoplasm which will neutralize a toxin (poison)

Apical. At the tip or point.

Apogamous. Displaying apogamy.

Apogamy. The formation of young plants from the vegetative tissue without either sperm or egg.

Apothecium. An ascocarp in which the hymenium lies exposed while the asci are maturing. The cup-like ascocarp of *Peziza* or *Sclerotium*.

Appendages. Processes of any kind which occur on bodies. Example: the appendages on the ascocarp of *Erysiphe polygoni*.

Appendiculate. Possessing appendages

Appresoria. A cup-shaped organ formed at the end of a hypha by which it is able to force a way through the tissues of a host plant.

Approximate. Near to or close together.

Arachnoid. Like a spider web. Cobwebby in appearance.

Arcuate. Curved.

Areolar. A space or pattern marked on the surface as in the case of some spores or seeds.

Aristate. Awned.

Ascigerous. Bearing asci.

Ascocarp. A fruiting body (fungal) that bears asci.

Ascogenous. Producing asci.

Ascogonium. The cell, or group of cells, fertilized by a single sex act and producing asci. It may result in the production of a perithecium, a cleistothecium or an apothecium.

Ascoma. The receptacle and hymenium of the larger ascospore bearing fungi.

Aseptate. Without septa or cross walls as in the case of the vegetative mycelium of the *Phycomycetes*.

Asexual. Vegetative. Without sex organs.

Asperate. Rough. Hairy or with hairy points.

Attenuate. Tapering to the tip.

Auriform. Shaped like a human ear.

Autecious. A condition in which the parasite spends its entire life cycle on a single host plant. Example, the rust of linseed.

Autogamy. When nuclei in the female cell fuse and cell fusion does not occur.

Automyxis. Following copulation of cells the nuclei may fuse with each other if they are lying adjacent to each other.

Autotrophic. Living on inorganic substances and the CO_2 of the air.

B

Bacillar (bacilliform). Rod-shaped or straight.

Basal. At the base of. Formed at the base or growing at the base.

Bacillus. A genus of the *Bacteriaceae.* A spore forming, rod-shaped organism. *B. subtilis* as the type species.

Bacteria. The fission fungi. The *Schizomycetes.* One-celled organisms without chlorophyll. Dividing by a simple fission.

Bactericide. A substance that will kill bacteria.

Bacteriophage. A living virus (so thought) which will cause the death of bacteria.

Basidiomycetes. A group of fungi characterized by a fruiting structure known as the basidium. The basidium bears typically four one-celled spores, basidiospores.

Basidiospore. The haploid spore produced on the basidium.

Basidium. The mother structure from which the basidiospores are formed. Found typically in the rusts and smuts among the plant disease fungi of the farm.

Biologic form. A physiologic race or a variety of a fungus.

Biotype. A single one of a group of individuals which are alike genetically.

Blastospore. A spore which has been budded off as in the case of of yeast cells.

Blight. A name commonly used to describe the condition in which there is a sudden death of the leaves and stems of plants.

Bordeaux-mixture. A spray (fungicide) consisting of copper, lime and water. A typical formula being 4℔s. Copper sulphate, 4℔s. lime (quick) and 50 gallons water. Some substance, such as casein, may be added to reduce the surface tension of the plant and act as a spreader.

Budding. A process of multiplication in one-celled fungi, or in the case of some of the higher fungi, in which new cells are cut off by pinching out a portion of the cytoplasm and a nucleus to form a secondary cell.

Bulbil. A small sclerotium-like structure composed of a number of cells.

Bulbous. Having a swelling at the base.

Bunt. In which the interior of the kernel becomes a mass of sooty spores. Usually associated with a fishy odour. Example: bunt of wheat.

C

Caecoma. An aecium without peridial cells and with or without paraphyses.

Calcareous. Like chalk in appearance. Chalky.

Callose. Being hard, thick or perhaps rough.

Calyculus. A cup or a calyx-like structure at the base of the sporangium in the case of some of the *Myxomycetes.*

Calyptra. A hood or cap-like structure on top of the sporophyte as in the case of some mosses.

Campanulate. Bell-shaped.

Canker. A condition in which there is a necrosis (death) of the tissues and the dead portions fall away leaving scars, usually sharply limited.

Capilliform. Hair-like.

Capillitium. Sterile thread-like fibres mixed with spores within a sporangium.

Capitate. Having a well formed head.

Capitellum. Having a little head.

Carbonaceous. Dark coloured. Composed mostly of substances in which carbon predominates.

Carpogonium. The female sex organ of algae or fungi.

Carpophore. The stalk of a sporocarp. The portion of the pedicel bearing the two halves of the fruit of the *Umbellifera.*

Cartilaginous. Like cartilage. Hard, tough or sometimes leathery.

Catenulate. In chains. End to end as the spores of the powdery mildews.

Caulicolous. Living on stems.

Cespitose. The growing together in tufts.

Chlamydospore. A thick-walled spore. A resting spore. Common in the smuts.

Chromogenic or chromogenous. Able to produce colour.

Ciliate. Possessing hair like locomotor organs.

Cilium. A process or appendage which is whip-like and capable of motion. It is an extrusion of living protoplasm and forms the locomotor organs of many of the protozoans.

Cinereous or cineraceous. Meaning ashy in appearance.

Circinate. Coiled into a ring-like structure.

Cirrhose. Possessing a cirrus or tendril.

Cirrus. A curl-like tuft or a tendril-like mass of spores which issues from a spore body. May be cloud-like.

Clathrate. Lattice or net-like.

Clavate. Club-shaped. Thickened toward the apex.

Clavulate. Somewhat club-shaped.

Cleistothecium. An ascocarp without an ostiole as in the case of the powdery mildews.

Clypeate. In the form of a buckler or shield. Possessing a clypeus.

Clypeus. A buckler or shield-shaped structure about the mouth of a perithecium.

Coadnate. United or joined together.

Coagulation. Formation of a clot and later separation of the clot from the serum as in blood, or the casein as in the case of milk.

Coalescent. Joining together.

Coccus. A spehrical bacterial cell. A genus of bacteria.

Cochleariform. Spoon-like.

Cochleate. Shell-like in form Twisted.

Coenocyte. A multinuclear cell.

Coenocytic. A multinuclear cell or mycelium without septa or cross walls. Example the *Phycomycetes.*

Collabent. Falling in or collapsing.

Colliculose. With little round elevations.

Columella. The sterile axile body within a sporangium.

Colony. A mass of individuals or vegetative growth representing the product of one or more individuals as in the case of bacteria, yeasts or other fungi.

Columnar. Column-like.

Comate. Hairy or shaggy.

Commensalism. A form of symbiosis.

Commissure. A seam. A place where a union has occurred.

Comose. Having hairs in tufts.

Compaginate. To be joined closely together.

Compliment. To complete. A substance found in the animal blood which aids in protection against disease producing organisms.

Complicate. Bent upon itself.

Compound. Similar parts aggregated into a common whole.

Concave. Hollowed out. Basin-like.

Con. A Latin prefix meaning to go with.

Conchate. Resembling one half of a clam shell.

Concolorous. Of one colour.

Concrete. Joined by growth.

Confluent. Blended into one.

Congested. Very near together. Crowded.

Conglobate. Collected into a ball.

Conidia. Plural of conidium.

Conidiophore. A hyphal thread bearing spores (conidia) at the tip or along the sides.

Conidiospore. A spore borne on a conidiophore. A conidium.

Conidium. A single spore (asexual) borne on a conidiophore. This definition does not include sporangiospores or chlamydospores.

Conjugate. To join together in twos.

Conjugation. Union of two gametes to form a zygote.

Connate. United by growth.

Constricted. To be contracted at one point. Drawn together at one place as a belt drawn tightly about the middle.

Continuous. Without septa.

Convolution. Convoluted. Rolled around the edge.

Copulation. The fusion of sex cells.

Coremium. A mass of spores that are broom-like in shape.

Coriaceous. Consisting of a leathery texture.

Corneous. Horny as to texture.

Cornute. Horned or horn-like.

Coronate. Crowned. Corona a crown.

Cortex. The outer coating of fungi. The peridium.

Cortical. Relating to the cortex.

Corticolous. Living on bark.

Costate. With veins or ribbs.

Cotyliform. Plate or wheel-like.

Crateriform. Goblet or cup-like.

Crenate. Tooth-edged.

Cristate. Crested.

Cruciate. Crust-like.

Crustaceous. Having a crust-like covering. Example the *Crustaceae* have a crust like outer shell or exoskeleton.

Cuboid. Like a cube.

Culture. A growth of bacteria or fungi used for study or experimentation.

Cumulate. Heaped up.

Cuneate. Wedge-shaped. Thin at one edge.

Cup Fungi. The *Discomycetes.*

Cupuliform. Cup-shaped.

Cuticle. The epidermis covering or outer most tissue covering a plant or animal body. In plants is composed of a waxy secretion.

Cuticulate. Having a cuticle.

Cylindric. Elongated, tubular or cylindrical.

Cyme. A cluster of fungus hyphae or spore masses with a determinate flattened top.

Cyst. A resting spore.

Cystidium. A sterile one-celled body, may be inflated, which projects beyond the basidia and paraphyses in the case of the hymenium of the *Agaricales.*

Cytolysis. Dissolving of the cell walls of fungi or bacteria. A term used to refer to the destruction of bacteria by chemicals and biological agents.

D

Dactyloid. Finger-like. Divided into projections like finger.

Damping-off. A rotting of the seedlings at the top of the soil. The plants develop a soft rot and fall over with the top and leaves still turgid and green. Caused by *Pythium debaryanum,* other *Pythium* spp. *Rhizoctonia, Slcerotium* and other fungi.

Deciduous. Referring to the loss of plant parts in season.

Declinate. To be bent or curved downward.

Decumbent. Bent over with the tip turned upward. Many trailing vines, lodged grain and other plants may assume this position.

Decurrent. Running down. Growing downward.

Dehiscent (dehiscence). A mode of opening when mature. Referring to the fruit. As a capsule.

Deliquescent. To become liquid. To dissolve.

Dematoid. Black. Cobwebby or spider web like.

Dendritic. Branching like the limbs of a tree.

Dendroid. Similar to above. Tree-like in form.

Dentate. Tooth-like. A leaf edge may be dentate when it has sharp tooth-like projections.

Denticulate. Minutely toothed.

Denuded. Uncovered. Exposed.

Depressed. Sunken. Flattened.

Dependent. Hanging down.

Determinate. A definite terminal point.

Di. Latin prefix meaning two.

Diaphonous. Transparent or nearly so.

Dichotomous. Forked or divided into two nearly equal parts.

Diclinous. Having the sex organs on different plants.

Difform. Irrigularly formed. Having a double form.

Dictyospore. A spore with cross walls at right angles to each other.

Didymospore. A two-celled spore.

Diffuse. Spreading widely scattered.

Digitate. Divided into finger-like projections.

Dimidiate. Divided into unequal halves. In the case one half appears completely wanting.

Dimorphic. Having two forms.

Dioecious. The sex organs being on separate plants.

Disciform. Flat and circular.

Discoid. Resembling a disc.

Disinfectant. A chemical or other substance which is toxic toward microorganisms.

Disjunctors. Spindle chaped connections between conidia.

Dissipiment. A partition.

Distichous. In two lines.

Diurnal. A cycle occurring daily or as a result of light.

Divaricate. Divergent.

Doliform. Barrel-shaped.

E

Ex. Referring to outer. From without. e.g. Exocarp meaning the outer part of a fruit.

Echinate. Having sharp pointed spines.

Echinulate. Having small spines or prickles.

Effuse. Expanded or spreading.

Elator. A slender thread-like hypha of the *Myxomycetes*.

Ellipsoid. Ellipsoidal Elliptic. All refer to an ellipse. Shaped like an ellipse.

Embedded. Sunken beneath the surface. Beneath. Surrounded.

Endo. Prefix meaning within or inside. Example. Endospore a spore within.

Endogenous. Produced within.

Endophyte. Growing inside another plant.

Endospore. A spore formed within. Endogenously.

Endozoic. Living within an animal.

Entire. Without a break. As an entire margin of a leaf.

Entomogenous. On insects.

Enzyme. An unorganized or soluble ferment. A biological catalyst.

Epi. A Greek word meaning upon. A prefix. Example. Epidermis.

Epidemic. Outbreak of disease in a group in one place, at one time.

Epiphytotic. An outbreak of disease upon plants.

Epiphyllous. Growing upon leaves.

Epispore. The outer coat of a spore.

Epithecium. The surface of a fruiting disc as in the case of the Discomycetes.

Epixylous. Growing on wood.

Erumpent. Breaking through.

Eu. A prefix meaning true. As in Eumycetes. Also when only a part of the thallus is used for developing the fruiting body.

Evanescent. Temporary. Soon disappearing.

Exospore. Outer covering. The wall of a spore.

Explanate. Spread out flat.

Exserted. Protruding beyond as in the case of stamens in some flowers.

F

Faciate. Massed or joined side by side.

Fascicle. A little bundle.

Faciculate. Growing in facicles.

Facultative. Sometimes; Not obligate.

Fastigiate. Growing as upright branches that are massed.

Favoid. Like a honey comb.

Fenestrate. With pores like windows.

Fermentation. A chemical change in organic compounds produced by ferments or enzymes.

Ferruginous. Rust coloured.

Fertilization. Union of sex nuclei.

Fibril. A very small fibre.

Fibrillose. Having fibres.

Fibrous. Made up of fibres or hyphae resembling fibres.

Filamentuous. Composed of threads.

Filiform. Thread-like in shape.

Fimbriate. The margin bordered by long slender processes.

Fission. Dividing by a simple splitting into two halves.

Flabelliform. Fan-shaped.

Flaccid. Flabby. Not turgid.

Flagellate. Possessing flagella.

Flagellum. A whip-like process possessed by single celled organisms by which they move about in a liquid medium.

Flavous. Nearly pure yellow.

Fleshy. Succulent.

Flexuous. Flexose. Bent in various directions. Capable of being bent.

Flocci. Groups or tufts of cotton-like growths.

Flocose. Cottony, bearing flocci.

Flocculent. The property of being aggregated together as in a bacterial culture.

Fluorescent. The property of giving off light under a radiation.

Foetid. Stinking.

Folicolous. Living on leaves.

Foliose. Leaf-like as in the case of lichens.

Free. Not attached.

Friable. Easily broken or powdered.

Fructicolous. Living on fruit.

Fructification. A fruit body.

Fruticolous. Living on shrubs.

Fruticose. Shrubby. Shrub-like.

Fugacious. Soon perishing.

Fuligineus. Fuligenous. Sooty or soot coloured.

Fulvous. Yellow, tawny.

Fumaginous. Smoky or sooty.

Fungicide. A substance used for the destruction of fungi.

Furcate. Forked.

Fuscous. Dusky. Brownish gray.

Fusiform. Thick but tapering towards each end.

Fusoid. Somewhat fusiform.

G

Gametangium. A structure which produces gametes. A differentiated cavity which produced gametes.

Gamete. A sexual cell.

Gangliform. Possessing knots or being knotted.

Gelatin. A colloidal substance made from bones and tissues of animals which will jell at certain temperatures and become liquid at others. Used as a food and as a medium for bacteriological culture work.

Gemma. A young bud. A vegetative propagating body as in the liverworts.

Gemmation. Budding.

Gemmiform. Bud-like.

Geniculate. Bent like a knee.

Genom. A haploid set of chromosomes.

Geophilous. Earth loving. Referring to the underground fruit bodies of some of the fungi.

Geotropism. The response to gravity.

Germicide. A substance having the power to destroy germs.

Germ pore. A pore or pit in the spore wall by which the germ tube may escape.

Gibbous. Relating to the shape of the pileus of some fungi. When the umbo is broad and the sides are short forming a flat under surface.

Glutinous. Resembling glutin. Sticky or mucilaginous in nature.

Gonoplasm. The central portion of the protoplasm in an antheridium which undergoes fission.

Gonosphere. A zoospore of the Chytridiales.

Gummosis. A disease of plants in which there is an exudate or gum that appears in cracks or breaks of the bark.

Guttate. Having a tear-drop-like shape.

Gyrate. With waves or furrows like the brain.

H

Haplo. A prefix meaning only one or only one half. Example. haploid meaning having n=chromosomes.

Hastate. Spear or arrow-head shaped.

Haustorium. A special organ or branch of a mycelium which serves as an organ of attachment and for absorption of food. As in the powdery mildews.

Helicospore. Rolled up or cork screw-like in shape.

Helminthoid. Worm-like in shape.

Hemi. A prefix meaning half or a part.

Herbicolous. Living on herbs.

Hetero. A prefix meaning different, other, as for example, heteroecious which means that there are two different stages of the fungus on different host plants. Black stem rust of wheat.

Hiascent. Becoming wide open.

Hilum. A scar on the seed where the funiculus was attached during the time it was in the fruit. Example the bean.

Hirsute. Possessing long hairs. Histogenesis. Produced from tissue.

Histolysis. To dissolve or to disappear.

Hoary. Gray from fine pubescence.

Holdfast. A structure which acts as a root to attach to an object.

Holo. A prefix meaning all, whole or entire. e.g. holocarpic in which all of the body used for the fruiting.

Homo. A prefix meaning the same or alike. e.g. homologous which is alike.

Host. A plant which nurishes a parasite.

Hyaline. Transparent.

Hydrotropism. Movement to water.

Hygrophanous. Having a water soaked appearance.

Hymenium. A layer of fungous tissue which produces the spores.

Hymenophore. That part which bears the hymenium.

Hyper. Meaning above. e.g. Hyperplasia is the over development of an organ.

Hypha. A single thread of a fungus.

Hyphal. Pertaining to hyha. Hyphoid. Hypha-like.

Hypo. A prefix meaning under or below or less. Ex. hypogyny. The condition in which the other parts are below the pistil in the same flower.

I

Imbricate. Partly covering one another as the tiles of a roof.

Immersed. Sunken beneath the surface.

Immune. Being free from or having the quality of preventing disease.

Imperforate. Absence of any openings.

Incrassation. A thickening of growth.

Indigenous. Natural to an area. Said to have originated in a country.

Indehiscent. Not opening along the suture lines.

Indeterminate. Not definitely determined.

Indurate. hardened.

Inermous. Absence of spines or prickles.

Inferior. Meaning ovary below the other parts in a flower.

Infundibuliform. Shaped like a funnel.

Innate. Immersed or to be embedded.

Inoculum. The culture or material used to produce the disease.

Inter. A prefix meaning between. e.g. intercellular meaning between cells.

Intra. A prefix meaning within. Inside. e.g. intracellular meaning within a cell.

Intramycelial. Within the mycelium.

Introrse. Meaning inward. In the direction of the central axis.

Intumescence. A swelling.

Invaginate. To cover. Invaginated. To be covered.

Involute. Having the edges rolled upward as the edge of the leaves.

Irpiciform. Toothed with the teeth resembling those of the fungus irpex.

Isabelline. A dirty tawny colour.

Iso. A prefix meaning alike or equal. e.g. isogamous the condition of having equal gametes.

Isogamy. The conjugation of two gametes of equal form.

L

Labium. Lipped as in the case of the mint flowers.
Labyrinthiform. Marked with lines that form a net or confused pattern.
Laccate. To be polished or shiny.
Lacerate. Torn or irregularly cleft.
Lacineate. As if cut into bands.
Lactiferous. Bearing latex as the rubber plant.
Lacuna. A hole or hollow.
Lageniform. Swollen at the base but narrowing towards the top.
Lamella. A gill or veil like structure. Lamellate. Made up of thin plates.
Lamelliform. In the shape of a plate or scale.
Lamelloid. Resembling lamellae.
Lanate. Wool like.
Lanceolate. Tapering toward each end.
Languid. Hanging down.
Latericious. Brick red in colour.
Latticed. Cross barred.
Lax. Loose. Separated.
Latex. A milky juice found in many plants. Example, the rubber plant.
Lenticular. Shaped like a double convex lens.
Leucosporus. Spores appear white when seen in mass.
Ligneous. Wood-like.
Ligulate. Flat and narrow.
Linear. Narrow. Several times longer than wide.
Linolate. Marked with lines.
Lituate. Forked with the points turning away from each other.
Longicollous. Having long beaks or necks.
Lobate. Divided into or bearing lobes.
Locule. Having cavities or cells.
Lophotrichiate. Having flagella at each end of the cell.
Lorate. Strap-like in shape.
Lumen. The space inside a wall as the central cavity of a cell.
Lunate. Shaped like a new moon.
Lysigenous. Formed by the breaking down of the cells.

M

Macro. A prefix meaning large or great. Example macrospore meaning the larger spore.

Maculicole. To be on spots.

Malacoid. Mucilage-like.

Mammiform. Breast or nipple-shaped. Marginate. Having well defined margin.

Matrix. The basis material from which a fungus grows as the body forming the mase of a lichen.

Medullary. Referring to the pith.

Mega. A prefix meaning large. Example megaspore.

Melanosporous. Black spored.

Melleus. Honey-like.

Membranous or membranaceous. Thin and semi-transparent like a fine membrane.

Meristogenous. Referring to pycnidia that are formed by the growth and division of a single hypha.

Merogamy. Copulation between special sex cells or gametes.

Meront. A daughter myxamoeba which is cut off by division of a parent myxamoeba.

Merosporangium. In the mucors it is a cylindrical outgrowth which is formed at the swollen end of a sporangiophore and which results in the formation of a chain-like series of sporangiospores.

Meta. Prefix meaning different or changed, e.g. metabiosis in which two organisms act one after the other. It also means between as in the case of metaxylem which is the portion in the centre.

Micro. A prefix meaning small. e.g. microspore meaning a small spore.

Microsporangium. A sporangium which produces microspores.

Mono. A prefix meaning one or single e.g. monohybrid meaning a cross involving a single pair of chromosomes.

Monosporic. Bearing a single spore.

Monostichous. In a single verticle row.

Monotrichous. Having flagella at only one end.

Mucilaginous. Like mucilage. Sticky when wet.

Mucose. Slimy.

Mluti. A prefix meaning many. e.g. multiseptate. Possessing many septa.

Muricate. Rough with hard excretions on the surface.

Muoronate. Pointed, ending in a sharp point.

Muriculate. With very small excretions on the surface.

Muriform. The cells arranged like bricks in the wall. With both longitudinal and transverse septa.

Muticous. Blunt. Without a point.

Mutualism. A form of symbiosis.

Mycelium. The vegetative portion of the thallus of fungi which is composed of many fine threads.
Mycogonous. Living on fungi or being derrived from fungi.
Mycotrophic. Having mycorrhiza.
Mycorrihiza. A fungus associated with a higher plant in a symbiotic relationship with the roots of the higher plant.
Mytiliform. Clam shell like in shape.
Myxamoeba. Swarm-cells with purely amoeboid creeping motion.

N

Napiform. Turnip-like in shape and form.
Necrophyte. A saprophyte. An organism living on a dead one.
Nidose. With an unpleasant odour.
Nodose. With knots or knobs.
Nodule. A small knob or rounded body.
Nomen. A prefix meaning name. e.g. nomenconservanda meaning the preservations of that name.

O

Ob. A prefix meaning opposite or the reverse.
Obese. Meaning fat to excess.
Obclavate. Widest near the base. Opposite of clavate.
Obligate. Essential. Necessary.
Oblong. Longer than wide.
Obsolete. Wanting or very rudimentary.
Obtuse. A figure with ends rounded or blunt.
Ochraceous. Ocher-coloured. That is yellow with a reddish tinge.
Olivaceous. Like an olive in colour.
Omnivorous. Diversified. Capable of attacking a large number of hosts.
Oogonium. Female sex organ containing one or more oospheres.
Oosphere. Naked mass of protoplasm which is the female sex cell and will become an oospore after fertilization.
Oogamy. A non-motile egg and a small motile sperm in the process of union.
Oogenesis. The development of the ovum after fertilization.
Opalescent. The property of giving off an iridescent light.
Operculate. Furnished with a lid.
Operculum. A small lid-like structure which covers a fruiting body as in the case of the pycnidia of *Operculella*.
Ostiolate. Possessing an ostiole or opening.
Ostiole. A small opening in a fungus fruiting body by which the spores escape.
Oval. Rounded to broadly elliptic in shape.

Ovoid. Like an egg in shape.

P

Pallid. Light coloured or pale.

Palmate. Lobed-like the fingers of the hand.

Pannose. Felt-like in texture.

Papilla. A small protuberance on the surface like nipple.

Papillate. Having small papillae over the surface.

Papilliform. Shaped like a papilla.

Papilloid. Resembling a small nipple in shape.

Paragynous. Having the antheridium at the side of the oogonium.

Paraphysate. With paraphyses.

Paraphyses. Sterile threads or filaments which are found in the fruiting structure of fungi like *Ascomycetes*.

Parasite. An organism living on a living organism.

Parathecium. The fruiting structure of the *Ascomycetes* containing the asci.

Patella. A small pan-shaped structure. Patella bone on the knee.

Patellate. Shaped like a patella.

Patelliform. Like a small dish that is circular and with a rim.

Pathogen. An organism capable of causing a disease.

Pathogenic. The condition of one organism being capable of causing a disease in another.

Pedicel. A supporting structure.

Pedicellate. Being borne on a pedicel.

Pedogamy. A false wall or tissue. Mature and immature cells of a fungus.

Pellicle. A small skin a delicate membrane covering the tissues.

Pellucid. Wholly or partly transparent.

Peltate. Shaped like a dish with the pedicel in the bottom as in the case of water plant leaves.

Periclinal. Curved so that the surfaces are parallel.

Peridium. The wall of a sporangium or other fruit body.

Perforate. Pierced and containing openings.

Periphysis. A sterile hair about the mouth of a pycnidium.

Periplasm. That portion of the protoplasm of the sex cells which does not join in fertilization.

Perithecium. The fruiting structure of the *Ascomycetes* in which the asci are borne.

Peritrichiate. With hairs from all parts of the surface. Example the bacterial cells with flagella on all parts.

Peronate. A covering on the lower part of the stipe as in the case of volva or veil.

Persistant. To remain after the other parts have gone.

Phototropism. Movement to light.

Phyllogenous. Growing upon leaves.

Physiologic race. (form) Alike morphological but unlike in physiologic reaction. Example. Physiologic races of black stem rust of wheat.

Pileate. Cap-like in form.

Pileiform. Pileus shaped.

Pileus. Like the cap of a mushroom or toadstool.

Pilose. Having a soft hairs.

Pionnotes. A slimy spore mass. A sporodochium of *Fusaria.*

Planose. Shaped like a plane.

Plano. Plano spore is a motile spore or zoospore.

Plaque. A clear area in media which has been discolourized by a bacterial colony.

Plasmodiocarp. A sporangium of the Myxogastre which is asymmetrical.

Plasmodium. The naked body of a fungus which exhibits amoeboid action.

Plasmogeny. Fusion between sex cells in a plasmodium.

Plectenchyma. A thick mass of tissue formed of twisted fungus hyphae.

Pleisporous. Possessing a large number of spores.

Pleomorphic. Possessing more than one form.

Pleurogenous. Formed on the sides.

Plexus. A net work.

Plicate. Folded into creases lengthwise.

Plurivorous. Attacking a number of hosts.

Polar. Referring to the poles.

Poly. A prefix meaning many. Example polymorphic, having many forms.

Polysporas. Many spored.

Poroid. Having many pores or openings.

Porose. Containing pores.

Prohybrid. A mycelium possessing additional mycelium form from hybridization.

Prolate. Extended in the direction of the poles.

Proto. A frefix meaning primitive. Example. Protozoa. The most primitive of the animal groups.

Proliferous. Bearing offshoots.

Promycelium. A shortlived germ tube which precedes the permanent mycelium.

Pseudo. A prefix meaning false or temporary.

Pubescent. With soft hairs.

Pulverulent. Powdered with dust-like material.

Pulvinate. Cushion-shaped.

Punctiform. In the shape of a point or dot.

Pustular. Like little blisters.

Pustule. A small blister or pimple.

Pustuliform. Having slight blister-like elevations over the surface.

Putrescent. Becoming rotten. Decayed.

Pycnidiophores. A fruiting structure bearing pycnidiospores.

Pycnidium. The fruiting structure of fungi bearing pycnidia.

Pycnidiospore. A spore borne in a pycnidium. A vegetative fruiting structure of the *Ascomycetes* and many of the *Fungi Imperfecti.*

Pycnium. The spermagonium as in the rusts.

Pycnosclerotia. Sclerotia bearing pycnidia.

Pycnosis. A condition in which a portion of the thallus becomes raised or arched with a hymenium forming underneath.

Pycnothecium. The fruiting body resulting from pycnosis.

Pyriform. Resembling a pear in shape.

R

Radiate. Spreading from a common centre.

Radicating. To spread like the roots of a plant.

Ramicole. Growing on branches. Ramose. Branched.

Rangiferoid. Branched like the horns of reindeer.

Receptacle. That part bearing the organs.

Reniform. Kidney-shaped.

Repetition. A germination by a new spore like the first one.

Restingspore. A spore which is thick walled or from other characters is capable of withstanding heat or cold or drying.

Resupinate. Without a pileus.

Reticulate. Netted. A net-like pattern.

Retrorse. Backwards.

Rhadagiose. Having deep cracks.

Rhizoid. A root-like structure of the black molds or Mucors.

Rhizomorph. A root-like fungus growth.

Rhodosporus. Having light red spores.

Rhomboidal. Approaching a rhombic form or outline.

Rhynchorosporous. Having beaked spores.

Rimose. Cracked.

Rivulose. Marked with lines like tiny rivers.

Rostrate. Possessing a beak like structure.

Rostrum. A structure like a beak.

Rufous. Reddish in colour.

Rugose. Covered with wrinkles.

S

Saccate. Bag-shaped.

Saprophyte. A plant living on dead material.

Sarcinaeform. Shaped like the bacteria of the genus *Sarcina.*

Scabrous. Rough to the touch.

Scissile. Referring to the formation of gills on the pileus of the toad stools.

Sclerotioid. A structure like a sclerotium.

Sclerotium. A mass of fungal cells in the dormant state. A compact mass.

Scolecospore. A long thread or worm-like spore.

Scopulte. Broom-like or brush-like.

Scorpioid. With the main axis crooked like the tail of a scorpion.

Scutate. Like a rounded shield.

Scutellum. A shield-shaped cover of the ascoma.

Scutiform. Buckle-shaped.

Septate. Divided by partitions.

Septum. A partition dividing parts of a body.

Seriate. In series.

Serrate. Edged like the teeth of a saw.

Sessile. Without a stalk or support.

Seta. A bristle-like body common to the acervuli of the genus Colletotrichum.

Setaceous. Bristle-like.

Setose. When bristles occur on bristles.

Setulose. Resembling a fine bristle.

Shield-shaped. Like the shield used by warriors.

Sigmoid. Like the letter Sigma in the Greek alphabet.

Simple. Not divided or compound. One unit.

Sinuous. With deep waves or ridges.

Soft rot. A decomposition of the tissues with the giving off of water and the formation of a soft mass with or without odour.

Sorus. A structure bearing numerous spores. Uredosorus.

Spatulate. Like a *kurpi* or a druggists spatula.

Species. A single kind of plant or animal. One of a number making up a genus.

Sperm. The male sex cell.

Spermagonium. A fruiting structure bearing male gametes.

Spermatium. The fruiting structure bearing sperms.

Sphaeroidal. Round like a sphere.

Spindleform. Like a spindle in shape.

Sporangiophore. The fungus structure which bears a sporangium.

Sporangium. The sac which bears the sporangiospores.

Spore. A single-cell capable of reproducing another spore or a mycelial thread.

Sporidesm. A spore ball made up of numerous spores remaining more or less attached together.

Sporidium. Asmall spore.

Sporocarp. A many celled body serving for the production of spores.

Sporodochium. A mass of spore bearing hyphae characteristic of the *Tuberculariaceae.*

Sporogenous. Producing spores.

Sporophore. A spore supporting structure or a spore producing structure.

Sporophyte. The vegetative stage of the plants. The diploid or asexual stage.

Squarrose. Rough with scales.

Stalagmoid. Like a tear drop.

Staling substances. Substances which are the result of the respiration of living forms and which are toxic to the organisms which produce them.

Starters. Cultures (pure or mixed) of organisms used to ferment organic materials and used to initiate a fermentation. Example, butter starters used in the dairy.

Stellate. Star-shaped with points.

Sterigma. A stalk bearing a spore as in the case of the germinating basidium which produces basidiospores on short stalks or sterigmata.

Stipe.. The stalk supporting the pileus of toad stool.

Stipitate. Having a stipe.

Stolon. A sucker or runner like stem.

Stoloniferous. Bearing stolons.

Stratose. In distinct layers.

Striate. Marked with fine longitudinal parallel lines.

Strigose. Bearing hairs that are stiff and sharp pointed.

Strome. A cushion like body which bears perithecia.

Stromatic. Referring to the stroma.

Stuffed. Solid or without partitions.

Stylospore. A spore borne on a filament.

Sub. A prefix meaning below, under or less. Example. Submargin meaning below the margin.

Subiculum. A felted or felt-like stratum of fungi.

Subulate. Awl-shaped.

Sulcate. Grooved or with furrows.

Superficial. On surface or very thin.

Suspensor. A club-shaped or conical shaped portion of a hyphal thread near a gamete. The gamete bearing hyphae of the genus *Rhizopus*.

Sympodium. A compound axis.

Syn. A prefix meaning to grow together. Example syncaryon. A diploid zygote nucleus.

Synergism. An association of two organisms acting together to produce an effect neither could do alone.

Syngamy. Isogamy between morphologically alike cells.

Synonym. Another name of the same organism which has become invalid or is a name having been used later in history.

T

Take-All. A disease of the roots of wheat caused by *Ophiobolus graminis*.

Taxonomy. The science of classification of living things.

T. D. P. Thermal death point.

Teleutospore. A resting spore of the basidomycetes.

Teleutosorus. A structure bearing teleutospores.

Telium. Same as teleutosorus.

Terete. Circular in transverse section.

Terrestrial. Living on the ground.

Ternate. In threes.

Tesselate. Marked with a mosaic design.

Tetra. A prefix meaning four. Example, tetraspore meaning four spore stage.

Thalloid. Like a thallus in shape or texture.

Thallus. A vegetative body having neither root, leaf or stem.

Theccum. The hymenius of the *Ascomycetes*.

Tomentose. Densely pubescent. The hairs being matted or felted.

Tortuous. A vegetative body bent or twisted in various directions.

Torulose. Bent irregularly often with portions that are swollen.

Temelliform. Gelatinous in nature.

Tremelloid. Jelly-like in substance or appearance.

Trichogyne. The receptive organ or female structure.

Trident. With three teeth.

Triquetrous. Three-edged.

Trophocyst. A hyphal swelling from which a sporangiophore is produced.

Tropism. A suffix meaning reaction. Example, hydrotropism the reaction of living organisms to water.

Truncate. Cut off at the end or blunt.

Tubercular. Having a shape like a tubercle or producing tubercles.

Tuberculate. Beset with knobby projections or excrescences.

Tubular. A cylinder-like body which is hollow.

Tumid. Swollen or inflated.

Tunica. A thin white membrane covering a portion of the fruiting body.

U

Umbellate. Having the inflorescence in umbels.

Umblicate. Navel-like or having a projection like the naval cord ending.

Umbo. A central swelling on top of the pileus.

Umbrinaceous. Umbrinous. Umbrinus. Colored like raw amber.

Uncinate. With hooks.

Ungulate. Having claws or hoofs.

Unguliform. Hoof-shaped.

Uni. A prefix meaning one or single. Example unipolar. One polar as in the possession of flagella by bacteria.

Urceolate. Pitcher-like, hollow with the mouth contracted urnlike.

Uredium. The sorus in which uredospores are formed.

Urticle. A bladder-like body seen in some of the fungi.

Utiform. Bag-like.

V

Vacuolate. Possessing valcuoles.

Valsoid. Resembling valsea in shape. A type of stromata in some of the *Sphaeriales*.

Valvate. With valves to control the openings.

Vegetative. Aseual. The growing portion of the body.

Veil. A thin envelope which covers the sporophore before it expands in a pileus.

Velutinous. Velvety because of a coating of soft hairs.

Venose. Having veins.

Ventricose. Swelling out at the middle or at one side.

Vermicular. Worm-shaped.

Vermiculate. Same as above.

Verruciform. Worty or shaped like a wart.

Verticillate. Arranged in a whorl.

Vesicle. A bladder-like sac.

Viable. Living. Having the power of life or producing life.

Villi. Long weak hairs.

Villous. Bearing villi.

Vinose. Wine coloured.

Virgate. Banded or streaked.

Virose. Poisonous.

Virulence. Severe as referring to a disease or ability to produce disease.

Viscid. Sticky.

Vittete. Having longitudinal bands or ridges.

Volva. A cup-like structure at the base of the stipe in the case of some of the *Agaricales*. The remnant of the veil which ruptures when the sporophore bursts through.

W

White rust. Rusts-like fungi of the genus *Cystopus* found on the crucifers.

Winter spore. A resting spore which carries the fungus over the winter weather.

Witches broom. A mass of outgrowth which is abnormal and produced by some fungus or abnormal condition. Many fine branches arising from a single point.

X

Xyloma. A sclerotium-like body that produces spore bearing tissue inside.

Z

Zonate. Marked into areas or zones.

Zoogloea. A mass of bacteria or of protozoans. Usually embeded in some matrix.

Zoosporangia. Sporangia that produce zoospores.

Zoospore. A motile spore. Usually the asexual motile spore.

Zygophore. The special branch in the case of *Rhizopus* which produces the gametes.

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ERRATA

Page viii, lines 14—16—*Omit* ‘To the Standard Agricultural Chemicals, Hoboken, New York, for the pictures shown in Chapter III.’

,, xiv, line 22—For ‘Wollen-waber’ *read* ‘Wollenweber’.

,, xiv, ,, 27—For ‘year’ *read* ‘decade’.

,, 1, ,, 21—For ‘of’ *read* “or’.

,, 7, lines 21—23—For the sentence beginning with ‘The *Phycomycetes* are often referred to . . . .’ *read* ‘Some *Phycomycetes* are referred’ to . . . .’

,, 10, line 21—For ‘zygote’ or ‘zygospore’ *read* ‘oospore’.

,, 10, ,, 26—For ‘zygospore’ *read*, oospore’.

,, 18, lines 9—14—Change the sentences to read ‘the *Ustilaginaceae* produce a septate basidium, the basidiospores being produced along the side of the basidium. The *Tilletiaceae* produce a non-septate basidium, the basidiospores being produced at the end of it in a cluster’.

,, 18, ,, 31 & 32—*Omit* ‘they may be within . . . . wheat’.

,, 22, ,, 4—9—*Delete* the words beginning with ‘In some of the rusts’ and ending with ‘said to be *heterothallic*’.

,, 24, line 20—For ‘vegetative’ *read* ‘asexual’.

,, 24, ,, 21—For ‘vegetative phases’ *read* ‘asexual reproduction’.

,, 29, ,, 15—For ‘spots’ *read* ‘spores’.

,, 30, lines 5 & 6—*Omit* ‘where found’.

,, 32, ,, 16 & 17—For ‘classifies’ *read* ‘studies’.

,, 32, line 18—*Omit* ‘Mycology is the oldest . . . . fungi study’.

,, 38, ,, 12—For ‘bi-flagella’ *read* ‘bi-flagellate’.

,, 41, ,, 31—For ‘gamete’ *read* ‘gametangium’.

,, 44, lines 2 & 5—For ‘zygospore’ *read* ‘oospore’.

,, 44, line 33—For ‘Zygotes’ *read* ‘Oospores’.

,, 47, ,, 29—For ‘Zygote’ *read* ‘Oospore’.

,, 53, ,, 4—For ‘radically’ *read* ‘radially’.

,, 55, ,, 7—For ‘radical’ *read* ‘radial’.

,, 55, ,, 20—For ‘on only’ *read* ‘or with’.

Page 58, line 14—For ‘that’ *read* ‘to’.
,, 68, ,, 25—For ‘Two’ *read* ‘Three’.
Add ‘Entyloma’ after ‘Urocystis’.
,, 73, ,, 6—For ‘*Hyalospermae*’ *read* ‘*Hyalosporae*’.
,, 73, ,, 7—For ‘*Phaeospermae*’ *read* ‘*Phaeosporae*’.
,, 77, ,, 29—For ‘imperfect’ *read* ‘perfect’.
,, 87, ,, 17—For ‘Kunkee’ *read* ‘Kunkel’.
,, 123, ,, 14—For ‘phtophthorus’ *read* ‘phytophthorus’.
,, 134, lines 14—17—For ‘Quarantine offices . . . . Tuticorin’ *read* ‘A quarantine office has been established in the Port of Bombay’.
,, 152, line 30—For ‘(*Urocystis occulta*)’ *read* ‘(*Puccinia glumarum*)’.
,, 159, ,, 23—*Add ‘Hordeum’ after ‘Triticum’.*
,, 162, ,, 14—For ‘Oogonium’ *read* ‘aecial initial’.
,, 162, ,, 21—For ‘trichigyne’ *read* ‘trichogyne’.
,, 162, ,, 21—For (‘Oogonium’) *read* ‘aecial initial’.
,, 162, ,, 12—For ‘Pycnidium’ *read* ‘pycnium’.
,, 164, ,, 15—*Add* ‘rust’ *after* ‘wheat’.
,, 166, ,, 20—For ‘*stritici*’ *read* ‘*tritici*’.
,, 170, ,, 5—For ‘epidemic’ *read* ‘epiphytotic’. (This change to be made in the remaining pages wherever the word ‘epidemic’ is used in the same sense).
,, 170, ,, 15—For ‘stormatal’ *read* ‘stomatal’.
,, 170, ,, 27—For ‘six’ *read* ‘seven’.
,, 177, lines 12—14—*Delete* ‘However . . . . Neovossia’.
,, 185, line 2—For ‘maize’ *read* ‘mays’.
,, 190, ,, 7—*Add after* ‘world’ (no full-stop) ‘but has not been fully demonstrated to be present in India’.
,, 197, ,, 32—For ‘covered’ *read* ‘loose’.
,, 198, lines 8 & 31—For ‘covered’ *read* ‘loose’.
,, 198, line 33—For ‘stop blotch’ *read* ‘Foot rot’.
,, 214, ,, 34—For ‘preudostromatic’ *read* ‘pseudostromatic’.
,, 216, ,, 27—For ‘Loose smut of Jowar’ *read* ‘Grain smut of Jowar’.
,, 217, ,, 24—For ‘The grain smut of Jowar’ *read* ‘The loose smut of Jowar’.
,, 224—The diagram should be opposite page 335.

Page 236, line 4—For 'common' *read* 'grain'.
,, 239, ,, 13—For '*turicicum*' *read* '*turcicum*'.
,, 243, ,, 7—For '*reilianum*' *read* '*reiliana*'.
,, 245, ,, 4—For 'oogonia' *read* 'aecial initials'.
,, 266, lines 7 & 8—For 'varieties' *read* 'symptoms'.
,, 273, line 3—*Delete* the words 'See photograph'.
,, 279, lines 9 & 10—For 'In this way . . . . planted' *read* 'In this way diseased tubers may be detected . . . . and planted'.
,, 281, line 24—For 'strong' *read* 'storage'.
,, 284, ,, 2—For '*Eubacteriaees*' *read* '*Eubacteriales*'.
,, 290, ,, 10—For 'that' *read* 'than'.
,, 296, lines 16 & 24—For 'conidia' *read* 'sporangia'.
,, 297, line 8—For 'egg' *read* 'oogonium'.
,, 305, ,, 28—For 'cellet' *read* 'celled'.
,, 305, ,, 13—*Add* 'and' *after* 'America' and *delete* 'and India'.
,, 319, ,, 13—For '*Colletorichum*' *read* '*Colletotrichum*'.
,, 323, ,, 31—For 'ret' *read* 'red'.
,, 339, ,, 12—For 'Bann' *read* 'Haan'.
,, 357, ,, 27—For '*Colletorrichum*' *read* '*Colletotrichum*'.
,, 359, ,, 7—For '*Urmyces*' *read* '*Uromyces*'.
,, 362, ,, 6—For 'fro' *read* 'from'.
,, 365, ,, 8—For 'fromed' *read* 'formed'.
,, 366, ,, 7—For 'agricultural' *read* 'Allahabad Agricultural'.
,, 378, ,, 9—For '*batacicola*' *read* '*bataticola*'.
,, 379, ,, 2—For 'are' *read* 'was'.
,, 381, ,, 35—For 'species' *read* 'diseases'.
,, 391, ,, 14—For '50 to 11' *read* '11 to 50'.
,, 399, ,, 7—For 'sells' *read* 'cells'.
,, 399, ,, 10—*Add* 'P.' *after* 'conidiophores of'.
,, 401, ,, 8—For 'Aecida' *read* 'Aecidia'.
,, 410, ,, 3—For 'necessary' *read* 'serious'.
,, 427, ,, 25—For '*Urocystic*' *read* '*Urocystis*'.
,, 433, ,, 1—For 'must' *read* most'.
,, 436, ,, 6—For 'Ggric' *read* 'Agric',
and for 'Stot' *read* 'State'.
,, 436, ,, 25—*Add* 'Poncirus' *after* 'Citrus'.

Page 439, line 10—*Add* 'are' *after* 'they'.
,, 442, ,, 12—For 'or' *read* 'of'.
,, 442, ,, 16—For '*sinenis*' *read* '*sinensis*'.
,, 444, ,, 20—For 'app' *read* 'spp'.
,, 445, ,, 35—For '*Colletorichum*' *read* '*Colletotrichum*'.
,, 449, ,, 23—For 'meloses' *read* 'melons'.
,, 455, ,, 10—For 'former' *read* 'farmer'.
,, 463, ,, 11—*Add* 'it' *after* 'that'.
,, 464, ,, 12—For 'the' *read* 'are'.
,, 466, lines 3 & 8—For '*conothecium*' *read* '*coniothecium*'.
,, 470, line 7—For 'Walves' *read* 'Wales'.
,, 472, ,, 19—*Delete* the word 'fruit'.
,, 475, ,, 11—For 'foll' *read* 'fall'.
,, 483, ,, 32—For '*Penicillum*' *read* '*Penicillium*'.
,, 486, ,, 3—For '*Boytrytis*' *read* '*Botrytis*'.
,, 489, ,, 21—For '*nectator*' *read* '*necator*'.
,, 495, ,, 7—*Delete* words 'See photograph'.
,, 498, ,, 15—For 'sot' *read* 'spot'.
,, 509, ,, 31—For '*Thea chinensis*' *read* '*Camelliathea*'.
,, 510, ,, 14—For '280.6—561.2' *read* '61.2 × 280.6 × 7.2 —11.2 etc.'
,, 525, ,, 18—For 'rom' *read* 'from'.
,, 525, ,, 23—For 'genera' *read* 'genus'.
,, 532, ,, 25—For '*Meliotus*' *read* '*Malilotus*'.
,, 533, ,, 22—For 'rom' *read* 'from'.
,, 540, ,, 34—For 'coco' *read* 'coconut'.
,, 551, Reference 132—For 'Cragie' *read* 'Craigie',
,, 583, Reference 604—For 'Fridirick' *read* 'Frederick'.
,, 585, Reference 632—For 'Qunajer' *read* 'Quanjer'.
,, 603, Reference 914—For '*Slerotinia*' *read* '*Sclerotinia*'.
,, 604, Reference 922—For 'Ustrup' *read* 'Ullstrup'.

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